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THE
LARYNGOSCOPE.

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NUTRITION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY*

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This is our eighth annual editorial on this subject. During this period we have offered the following ideas and suggestions: (1) From the beginning we have felt that no vitamin can be expected to improve a pathologic condition which has not been caused by lack of that same vitamin—"no deficiency, no cure." (2) The embryologic approach to therapy; it appears that a special relationship exists between vitamin and germ layer; it seems that each germ layer demands an oil-soluble and a water-soluble vitamin; the ectoderm and entoderm seem to have a common need in that they demand the same vitamins—"A" and B Complex; and that the mesoderm requires "C" for the development of connective tissue and "D" for the development of bony tissue. If this concept is correct, we then have a rational approach to therapy. Throughout life the derivatives of each germ layer continue to demand the same vitamins that meet its special needs. (3) Rat experiments[1], [2]. Control rats received full diet. Experimental rats received the same diet except the B Complex; it soon became apparent that rats cannot even live without the B Complex, so a small amount was given in order to keep them alive. Histologic studies by Walter P. Covell[3], [4] showed that depriving rats of the B Complex produced no significant changes in the turbinates, tongue, larynx and trachea, but marked changes in the cochlea. The histologic appearance made it evident that these rats had a

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marked nerve deafness. (4) During the war when there were thousands of applicants for aviation, claims were made by optometrists and psychologists that defects in color vision could be cured by giving vitamin A. This "news" was broadcast over the radio throughout the country, and not only in popular but in scientific journals. We showed this to be wrong; we can continue to say to the aviator, "You are born with color vision—or without it." (5) Before the discovery of vitamins the notions of metabolism were foggy. We were aware of carbohydrates, proteins, fats, minerals and water, but we had no conception how they become transformed into living tissue. We now realize that raw materials and their products are valueless without the "activators"—the keys that open the door into the cell. There are two classes of activators—the intrinsic and the extrinsic. Those from within the body are the endocrines; those from without are the vitamins. (6) All physicians are confronted daily with problems due wholly or in part to a lack of the right foods. The tissues of the body which are most easily *observed* are those of the skin, eye, ear, nose and throat. Obviously, in detecting evidence of a deficiency, no one is in a better position than the Ophthalmologist and Otolaryngologist.

In our attempts to outline a concept of Nutrition, we subscribe to the spirit of a sign over a modest shop. "We think we do good work. We may be right." The work of Mickey[5] is not only superb but modest—he says that readers may regard his book as presumptuous; if they do he will not dispute them, for he regards it in that light himself. The book attempts to trace the relationship between the soil and human health, no less! This involves the gigantic natural cycle which includes the plant, the animal and man. It is hard to realize how recently Pasteur, by formulating his concept that bacteria cause disease, opened the gate leading to modern medicine. Today's sulpha drugs and penicillin are milestones along this road. The new ability to cope with infectious diseases at first was hailed as presaging a wonder-

ful new world of supermen, living in unalloyed happiness and health. This dream has failed to materialize. Although every infectious illness except the common cold is on the decline, the sum total of human disease has increased and continues to increase. Another sort of ill—degenerative disease —increasingly assails civilized man. Deterioration of body tissues results in dental caries, rickets, osteomalacia, arthritis, nephritis, arteriosclerosis and many other troubles. In these, bacteria play no part—or at most, a secondary part. Dental caries and periodontal disease are almost universally prevalent in modern civilization. Price[4] found dental caries practically non-existent among primitive peoples isolated from civilization. He also found that, even in the first generation after primitive races adopt the foods of white civilization, tooth decay appears; and with tooth decay, such evolutionary degenerations as narrowing of the face and dental arches. Disease of these structures is a measure of physical degeneration; it is almost invariably accompanied by deterioration of other tissues. Primitive races are capable of sustained physical effort that seems prodigious to us. The Indian Hunza frequently travels 60 miles on foot in one stage and makes his return trip at once; the Australian aborigines, the Esquimaux and many other primitive peoples are endowed with physical and nervous stamina far surpassing that of our most rugged and highly trained athletes. Leaders in the medical profession and allied scientific branches today regard degenerative disease as civilization's major health problem. Even making most generous allowances for advantages in the art of diagnosis, degenerative disease is on the increase. As to the cause, the trail has been blazed by the Cheshire County group in England, Sir Robert McCarrison in India, and Dr. Weston A. Price in the United States. In our country it was the dentist who led; and, in the awareness and practice of nutrition, it has been the dentist who through the years has been far out in advance of the Ophthalmologist and the Otolaryngoloist. After 25 years of study the Chesire group attributes the alarming increase in illness to "a life-time of wrong nutrition." McCarrison fed rats the same

diet as that eaten by peoples in various parts of India and produced in the animals conditions corresponding to what had appeared in the people. Price studied primitive peoples in many parts of the world and compared those living in isolation with their blood brothers exposed to white civilization. The situation presents a paradox: In the past century and a half the peoples of North America and Western Europe have had more to eat than any peoples ever had—yet within that very period physical degeneration has been on the increase. Prior to 1800, when hunger was the daily companion of the average man, degenerative disease appears to have been rare. It seems that man may be poorly nourished where and when his food supply is most generous. Peoples living at the lowest economic level—primitive hunters and fishermen—show the least evidence of degeneration, except when they come into contact with civilization. Pastoral peoples, on the next higher level, show relative freedom from dental decay and other degenerative conditions. Food is so scarce with these primitive hunters and shepherds that they eat all that is available; nothing goes to waste. They cannot afford, like peoples on a higher economic and cultural plane, to eat only the muscles of an animal and scorn the internal organs. As the latter are especially rich in the minerals and vitamins essential to health, the savage is likely to be healthier than man on a higher cultural plane. It is when he moves upward to an artificial existence, when he domesticates plants and practices agriculture, that physical degeneration begins. Finally, when life becomes urban and industrial, the degeneration becomes appalling. With a plentiful supply of food, civilized man is able to pick and choose. Lacking the instinct of the lower animals and the intuition and tribal lore of primitive man, he all too frequently chooses what is not good for him.

A chemist, Maynard[7], regards Lavoisier as the father of the Science of Nutrition. Lavoisier stated, 150 years ago: "La vie est une fonction chimique." Since that time chemistry has played the central role in developing knowledge of nutri-

tion. In 1912 the existence of the first vitamin was established—as a something, a quality, a property or perhaps a substance, which some foods had and others had not. It was 15 years before the chemical nature of any vitamin was definitely determined. We have come to know that thiamin is essential for the removal of pyruvic acid—an intermediate in carbohydrate breakdown; thus we can relate the requirement of thiamin to the intensity of energy metabolism. In the case of "D" however, although its chemistry has been known for years, we can make only the general statement that it is essential to calcium and phosphorus metabolism. How or when it acts we do not know. The same holds for other nutrients, both vitamins and minerals. Our understanding of nutritional requirements for the latter half of life is much less than for the period of growth, because we know less about the tissue changes which are taking place. A child falls down, jumps up and goes blithely on; an old person trips and breaks his hip. We understand the role of nutrition in building up bone during growth, but little about its possible contribution in retarding changes which come with age. Many findings in animals require testing for their human applications. It has been found that 0.1 mg. of cobalt daily spells the difference between life and death in the sheep; lack of this minute amount was responsible for the death of tens of thousands of animals yearly before this discovery was made. Cobalt is probably unimportant in human nutrition, but we are not sure about this. In many respects much more is known about feeding animals than feeding people. The "purified diet" procedure has proved one of the most effective methods in modern research; its early use with rats and dogs has now been extended to several other species, including man. Amino acids are coming into increasing use in purified diet as a method of determining their quantitative requirements.

Although the teaching of nutrition[5], [6] has always been an integral part of Pediatrics, such teaching was largely empirical until the past two decades. It is now based firmly upon knowledge. For example, knowledge of the rate of growth

of the skeleton at various stages during infancy and childhood is of real value, indicating calcium requirement at a given age; the treatment of severe diarrhoea requires knowledge of salt and water metabolism. Only recently has it been learned that it is as importnat to supply lost potassium as it is to replace sodium and chloride, if a baby is to recover from diarrhoea. Knowledge of the proper use of protein hydrolysates is relatively new, and in fact still incomplete. In animals the production of healthy offspring depends largely on the food consumed by the mother. Antepartum food influences the offspring's ability to grow and keep well. As a striking corollary, specific congenital malformations can be produced, almost at will, by feeding the mother foods deficient in certain essential nutrients. This is an old story in animal husbandry; but only in recent years has the physician come to realize its full significance in human terms. Medical students are taught about essential nutrients but little about how to utilize them. Frequently, even in the best fed areas of the country, pregnant women lack essential nutrients —particularly proteins, calcium, iron and "C".

In their thoughtful and thought-provoking book "Hidden Hunger," Macy and Williams[10] make us realize that the Science of Nutrition came of age in the twenty-one years between the World Wars. Consequently, during the second war, nutrition has played a major role—on the firing line and on the production line. In the tomorrows, Nutrition can be the greatest single factor in perpetuating the peace. Nutrition is the key to buoyant health—mental as well as physical. Whether or not the potentials of nutrition are to be fully realized remains to be seen. In this respect man's destiny can be self-determined. In the fifth century, B.C., Hippocrates prescribed a mode of living to prevent disease, and foods to cure or help specific ills; his emphatic writings on food, fresh air and sunshine constitute the earliest recognition of the importance of what we now term "Nutrition." He even sensed today's B.M.R.—"growing bodies have the most innate heat; they therefore require the most food. * * * In old people the heat is feeble and they require little fuel * * *"

One modern assay of the average adult man shows: enough water to fill a 10 gallon keg; enough fat for 7 bars of soap; carbon for 9,000 lead pencils; phosphorus to make 220 match heads; magnesium for one dose of salts; iron to make 1 medium sized nail; sufficient lime to whitewash a chicken coop; and sulphur enough to rid one dog of fleas. Another assay shows that if a young chemist should make an analysis of his girl friend he would find the ingredients of "Sugar and Spice and everything nice" could be purchased at the corner drug store for less than a dollar. The human body is about 5 per cent minerals—nearly 8 lbs. in a 154-lb. man. It must be remembered that these constituent particles are not fixed but are in continuous metabolic flux—as environment is converted into organism and organism into environment. Iodized salt has been marketed for over 20 years; about 50 per cent of all table salt is iodized. Few measures have been so successful in improving the public health as this initial step in food fortification. It is important that the diet of the pregnant woman include enough iodine to meet the needs of her own body and also those of her unborn child. Neglect of this results in cretinism. About 90 per cent of all margarine now sold is fortified with "A." English law required that all margarine be fortified with "A" and "D." The value of "restored" or "enriched" white flour has proven higher than had been expected; but it remains lower than that of unrefined flour. Today's problem is up to the housewife—what she demands the miller and baker will supply. Purchasers of available flour are too often concerned with expense rather than with value. Whole grain flour should be used in preference to refined white, even though the latter has been "enriched." Whole wheat is the "natural" food and contains all the elements originally present in wheat. Many people designate as "whole-wheat bread" a product which is perhaps 25 per cent whole-wheat flour. If the remaining white flour (75 per cent) is not enriched, these loaves offer fewer vitamins and minerals than the "enriched" white loaves. In the interval between the two Wars this infant bud, Nutrition, blossomed into full flower—of adolescence. Today we have no fear of wheat scarcity. Our first concern is quality. It is important to

satisfy the hidden hunger as well as the hollow hunger. The deficiencies of the past were not human errors; they resulted from lack of knowledge—but today we have that knowledge. One can find no better example of the strides made than in this matter of bread. Ironically, in the first War we thought we were patriotic in skimping ourselves on white flour and bread on the home front so that no soldier should have to eat the so-called "black bread." We shipped to the fighting front the deficient white bread and flour and soothed the injury to our sophisticated appetites with the salve of eating the more nutritious "black-bread." Now the dark or whole grain brands are of course recommended, although one may indulge a perverted palate and still obtain some of the nutrients lost through milling by eating the "enriched," "fortified," or "restored" white bread. Sugar is no longer recommended; the indulgence of the American sweet tooth has become a national scandal. The Greeks were noted for their teachings, but "moderation in all things" was apparently not translated into our language. We eat nearly twice as much sugar as any other people; it accounts for about 1/7th of our total food calories. We have gone along blithely crowding essential nutrients out of our diet by overconsumption of sweets, ignoring the danger to our teeth—and waistline. At the start of War II it was imperative to utilize all that was known about nutrition and to devise new methods. Under such conditions more was accomplished than in ten years of peace. For years Germany had realized the importance of nutrition to the soldier and to the worker; for every fighter eight workers were engaged in producing and handling food. For twenty years Japanese scientists had been compounding concentrated foods for their armies in preparation for aggression. England, fighting for existence long before we entered the War, gave us valuable information. We found that 39 per cent of our American workers were using good food, 26 per cent poor, 35 per cent only fair. The Royal Air Force, alarmed by the great number of applicants turned down as physically unfit, set up a depot for would-be recruits. They fed them properly—and salvaged 87 per cent of 834 men so treated.

Grain products contribute a varying, but very important part of food in nearly every land. In this country bread, flour and cereals furnished from 19 per cent to 54 per cent of the diet; in the Orient the proportion is much higher, up to 80 per cent or 90 per cent. Westerman and Hall[11] have attacked the problem of vitamin poverty of milled flour; 50 per cent of pyridoxine, for example, is lost in the milling process; the amounts of "B_1", "B_2", and calcium pantothenate are reduced 4.1, 1.1 and 7.7 mg. per gm., respectively. The albino rats used in these investigations were weighed every six days throughout a test period of 72 days. The rats fed on a B Complex-free diet died in about 18 days; those on ground whole wheat gained, on an average, 27 gm. during the test; those on enriched flour, an average of 30 gm. These investigations showed that the addition of 1 mg. of "B_2" per gm. caused an increase in the growth-rate; a further addition of 1 mg. of "B_1" stimulated even greater growth; calcium pantothenate in a proportion of 20.5 mg. per gm. and pyridoxine 7 mg. per gm. added to the other vitamins produced the best increase in growth. "He that filches from me my good name robs me of that which not enriches him and leaves me poor indeed." It is a pity that it is modern milling itself which robs flour of its natural rich content. One step in milling is to impoverish flour; another more recent step is to "enrich" by restoring, at least in part, what has been removed. The millers and bakers follow the public demand for white flour; but in reality the situation is like a housing-shortage problem in New York[12]. Thousands of ready-cut homes can be shipped in, but the law requires that the local carpenters knock the ready-cut walls apart and put them together again, that the local plumbers rip out the built-in plumbing and then put it back again, and so on—ad absurdum.

The principal opposition to so-called "enrichment" of flour and bread has come from the perfectionists. Williams[13] considers that this group, including many nutritionists, physiologists and bio-chemists of high standing, argues with formal logic that the proper correction is to use the whole grain

in the first place. Their number is not large and for some reason is gradually diminishing. If one were feeding dumb animals which are without means of effective protest, this might well be the answer. "Dumb" animals which are not without means of effective protest present quite a different problem; humans will eat what they like and complain if deprived of it. Ninety-eight percent of flour sold in the United States is white; substantial alteration of this will require many years, and more persuasion than the nutritionists of the country can muster. In our country about 75 percent of white bread and family flour has been enriched for the past three years or more. The British did an amazingly good job of sustaining the nutritional adequacy of their bread during the War. It was very monotonous and unattractive—and still remains so. Tisdall tells of the avidity with which Englishmen embarking for the United States attacked the "tasty white bread" offered on shipboard. In 1941 the Food and Nutrition Board of our National Research Council declared itself in favor of the following additions: "A" to oleomargarine, "D" to milk, iodine to salt, and thiamin, riboflavin, nicotinic acid and iron to white bread, white flour and corn meal.

In a careful study of 183 boys in a children's institution a high incidence of nutritional deficiencies was found by Biskind and Williams[11]. About 90 per cent showed glossitis, 67 percent gingivitis, 19 percent cheilosis, 60 percent nervous symptoms. The lesions were recorded by color photography under standard conditions of exposure and lighting—before and after treatment. The childrens' food was then fortified with B Complex, "C", "A", "D" and "E"; also calcium, phosphorus, iron, copper, iodine, manganese, magnesium and zinc. After seven months there was complete disappearance of the lesions in most of the boys and improvement in all but a few of the others. Nervous symptoms showed definite improvement in nearly all of the subjects. This study supports the view that search for signs of nutritional deficiency should have high priority. Recognition of such lesions on casual examination—mild lesions of the tongue, for instance—is

important; all too frequently these are apt to be passed over as unimportant. Similarly, observation of the eye may reveal latent deficiencies in nutrition. Members of the Marine and Naval personnel who served in the South Pacific islands for periods of 6 to 18 months came under scrutiny by Knapp[15] for various eye complaints. They were found to suffer from axial myopia, night blindness, and vernal catarrh; in addition, changes were found on ophthalmoscopy in certain of the service men—haziness of the fundus, pallor of the optic nerve, and in a few a minor degree of loss of pigment. The factor common in all patients was diet; canned food had been used almost exclusively with resulting deficiencies of vitamins and minerals. On the richer hospital diet improvement was prompt in the cases of vernal catarrh, optic nuritis and chorioretinitis. Of the men in whom axial myopia was found and believed to be of recent development, more than one-half improved from the good food and the use of "D" and calcium; either the degree of myopia was lessened (in 33 percent), or the condition remained stationary. It is of especial interest that, apart from these eye signs, the men did not show evidence of poor nutrition.

Impaired nutrition was found to be the essential cause of reduction in visual acuity in seventy-four school children in Kingston, Jamaica. In all the cases Whitbourne[16] found great difficutly in obtaining a satisfactory history of onset; in most cases the loss of vision was far advanced when the condition was recognized. visual acuity being 6/24 to 6/60, or less. Many of the children showed other evidences of under-nourishment, such as angular stomatitis and glossitis; deafness was the most serious accompanying defect. Milk and eggs were ordered, but in the majority of the cases these good foods were not available. Cod-liver oil and brewers yeast, with additional amounts of "B_1" and "B_2" in some cases, were given in varying quantities. In spite of economic difficulties fifty patients made satisfactory progress. Mouth lesions invariably cleared first; the visual and auditory impairments improved later. In long-standing cases the vision did not show the hoped-for gain.

The tragic story of malnutrition of prisoners in Japanese hands is repeated by Nardini.[17] Owing to confiscation of his papers by the Japanese he is unable to give statistics, but the concise recounting of suffering is more illuminating for the very lack of tabultions. Beri beri brought in its train neuritis of the optic and acoustic nerves; treatment was irregular, rarely adequate and, in cases of optic atrophy, wholly ineffective. The loss of hearing function was less marked. The toll of pellagra deaths attributed to cerebral changes, with dementia and coma, goes far to confirm the opinion of pellegra students in the southern states, such as is set forth in the report of Cleckley, Sydenstricker, and Geeslin[18]; in their cases nicotinic acid in high dosages proved to be life-saving. Other avitaminoses, due to "A" and "C" deficiencies, were the cause of much suffering. Lack of "A" resulted in xerophthalmia, and means of treatment were almost entirely lacking until the later periods of imprisonment. Scurvy from lack of "C" was surprisingly infrequent as a distinctive entity, but undoubtedly contributed to many deaths.

Experiments with rats were made by McCoy, Maynard, Sperling and Osgood[19] to seek light on nutritional requirements during the latter half of life. Little is known about the optimal diet after middle age and yet it is no less important than in the earlier years. The protein level of diet during the latter half of life was found not to affect the life span when protein constitutes between 8 and 30 per cent of the diet. When larger amounts of protein are taken, the non-protein nitrogen of the blood increases and the heart and kidneys become larger. The albumin of the urine is roughly correlated with the level of protein intake. Rats keep underweight by caloric restriction outlive those allowed to fatten. Exercise is of minor importance in comparision with body-fatness. The optimal conditions for long life proved to be thin bodies, exercise and low protein diet, with this protein provided by liver.

In the study of cancer, the main emphasis has been placed on genetics, the influence of hormones, viruses and specific

toxins— and other etiologic concepts. Usually nutrition is not included. Nevertheless, Rusch and Baumann[20] note that the behavior of living forms can be modified by foods—in practically all phases of the life cycle. Consequently it seems logical that the question should be raised whether development of abnormal tissue might also be modified by foods. Certain neoplasms are influenced by the kind or the amount of food. Claims to this effect are not new but advances in the science of nutrition have made it possible to devise experiments on tumor-bearing animals with results which have been again attained on repeating the experiment. For example, it is possible to retard tumor-formation by a 30 to 40 per cent reduction of the caloric intake of the animal. This lowering of intake usually results in temporary loss of weight; then bodyweight becomes stabilized at a somewhat lower level than that of fully fed animals. The development of tumors is largely suppressed—entirely, in some cases. The effects of food restriction are more marked upon the tumors than upon the rest of the animal. These effects of caloric reduction are not permanent and when full caloric intake is resumed tumor growth begins anew. The bearing upon the formation of human cancer is suggested by insurance statistics; the proportion of persons dying from cancer is greatest among those classified as "overweight," and it is somewhat greater for those average weight than for those classified as "underweight." In the rat, underfeeding not only prolongs life but also prolongs certain characteristics of youth. Indeed, experiments on tumor-bearing animals often suggest that "improvements" in the diet, such as increasing the "B" vitamins and protein "improves" the development and growth of the tumor and hence, hastens the animal's death.

PROTEINS

An amino acid is to protein what glucose is to starch. As Elman[21] states they are the unit components. The discovery, purification and identification of amino acids constitute one of the milestones in the development of the science of nutrition. The isolation from foods of these specific chemical com-

ponents represents the most important recent contribution of chemistry to our knowledge of nutrition. Unlike vitamins, amino acids actually enter into the material of body tissue. While they are used up like glucose, unlike the latter they are not burned up as fuel—unless calorigenic elements are lacking. They may act for glucose when it is unavailable. They are also unlike other purified food essentials in that they are so numerous. The glucose molecule represents the form in which carbohydrate is utilized. The B Complex has been subdivided into a number of crystalline substances; there are twenty-one or more amino acids, ten of which are deemed indispensable. The body is unable to manufacture them and they must be supplied before normal protein metabolism can take place. Normal growth takes place if these ten amino acids are fed to young rats as their sole source of protein; if even one of these is omitted, normal growth does not occur. In this sense these ten amino acids assume importance comparable with that of vitamins in maintaining life processes. By using amino acids instead of food proteins the physician can administer protein nutriment in its simplest form, just as he uses glucose. Giving amino acids by needle, he does not have to depend on the gastrointestinal tract. Like glucose and vitamins, amino acids are available in crystalline form and may be purchased on the open market. The greatest drawback to all this is the cost; but future methods may cheapen crystalline amino acids sufficiently to permit their general use. (The first few kilograms of synthetic thiamin cost ten dollars per gram. Recently, annual production exceeded fifty tons and the cost fell to sixteen cents per gram.) The oral preparations of amino acids require no protein digestion. In patients in whom ingestion of protein food provokes diarrhoea or vomiting, substitution of amino acids avoids such troubles. Amino acids may be taken by mouth dissolved in water, milk or juices. Vitamins must be given along with amino acids. The addition of amino acids to the usual parenteral fluids makes possible the administration of almost complete nourishment without digestive process. Thus, starvation can be avoided even though a patient be unable to take anything

by mouth. Complete rest of the gastrointestinal tract, which heretofore inevitably resulted in protein starvation, can now be achieved with an almost complete nutritional intake.

As to protein requirements for the average adult, Stare and Thorn[22] recommend 70 gm. protein daily. Many variations are possible, with the help of food composition tables and the application of nutritional and common sense. They suggest, for example, milk 1 pt. (15 gm. protein), cooked meat 3 oz. (18 gm.), 1 egg (6 gm.), peas or beans 3 oz. (2 gm.), other vegetables (3 gm.), fruits (2 gm.), bread, 6 slices (18 gm.). Such a daily intake gives 70 gm. of protein. It is of interest that 1 pint of milk contains almost as much protein as 3 oz. of cooked meat or 6 slices of bread. If the patient cannot take sufficient protein in the form of foods, concentrates may be added, such as corn or wheat germ, soy bean flour, dried yeast or peanut flour. Often it is difficult for the patient to consume an adequate diet, and it may be temporarily impossible for certain patients to swallow food. Tube feeding is then available. Parenteral protein therapy consists essentially in the intravenous administration of blood, plasma, albumin or various casein hydrolysates. In the rat, human plasma and albumin are inadequate when used as the sole source of protein for growth. They are satisfactory if isolucine is added to the plasma, and if isolucine and tryptophane are added to the albumin. Whether these observations apply in human subjects is not known, but they deserve consideration. Mixtures of amino acids are well utilized whether given intravenously or orally. When purified amino acids become available in sufficient amounts they should offer an ideal solution to the problem of intravenous protein therapy.

In the cases of 11 infants, 3 moderately ill and 8 well, Gluck and Wilson[23] gave diet formulas based on Amigen (50 per cent and Dextri-Maltose (50 per cent). The purpose was to study the babies' tolerance to large caloric intake of monosaccharides and casean hydrolysates. The babies' ages varied from 4 to 22 weeks. There were 18 trial periods; in 8 of these, sudden elevations of temperature were observed

ranging from 101 to 106°F—not explained by infection; in 7 there was fever attributed to known infections; in 3 the temperatures remained normal. Gains in weight were inconstant; in some the increased weight was lost after a return to regular milk formulas; in others it was maintained. Careful study of all influences linked the fever with the amino acid; but this febrile response remains obscure.

MINERALS

The minerals of the body are of tremendous importance. Because of the great interest in vitamins Harding[24] considers that too little notice has been taken of minerals; they have been pushed into a dark corner, like the proteins. However, the use of isotopes is focusing attention upon the minerals, which will probably soon receive the attention they deserve. The animal body must have seven essential minerals: calcium, magnesium, sodium, potassium, phosphorus, sulphur, chlorine. There are others of less importance. Calcium is present in larger amounts than any other mineral; about 99 per cent of it is in the bones and teeth. The relation of calcium to phosphate-magnesium-acid excretion as well as to the hormones and vitamins is most intricate. Calcium is better absorbed when one is on a high protein diet. About 80 per cent of the body's phosphorus is in the bones and teeth; half the remainder is present in combination with proteins, lipids, carbohydrates and other organic compounds in muscle. The calcium-phosphorus ratio is of great importance. When either is taken in excess the other is simply excreted over and above a certain minimum. The average ratio appears to be 1.1; but there must also be adequate "D" if the bones and teeth are to be normally formed and maintained. Recent research with radioactive phosphorus indicates that it can be readily absorbed in resistant rickets, provided renal function is unimpaired. The average adult requires 115 times as much phosphorus as calcium. Three-fourths of the body's magnesium is combined with calcium and phosphorus in complex bone salts. Little is known of the magnesium requirement. Sodium forms the largest frac-

tion of the total base in blood plasma and extracellular fluids—largely associated with chloride and bicarbonate ions. With them, sodium serves to maintain osmotic pressure and ionic equilibrum of body fluids and tissues. Human requirement of sodium is small. Rats maintained on low potassium diet showed necrosis of myocardial fibres and renal tubular epithelium; addition of potassium to this diet prevented these changes. The requirement for man is unknown; but, as all natural foods are good sources of potassium, it may be ignored. About 0.1 per cent of the body is chlorine. Human diet contains sufficient chlorine except during excessive losses due to diarrhoea or sweating. All the cells of the body contain sulphur. Only small amounts of inorganic sulphur occur in the blood and tissues, but it is a constituent of sulfatides, glutathione, insulin, biotin and thiamin. Hens store sulphur to make egg-yolk during periods of low production and use it when they go into high production. Of course, iron is an essential body mineral but the healthy male adult and the healthy woman after menopause, have very low iron requirements. The chief function of iron is the transportation of oxygen via hemoglobin. Iodine is essential to thyroid function. Its only known use is in the formation of thyroxin. It appears that copper is important because it is essential to the regeneration of hemoglobin. It is not a constituent of the hemoglobin molecule but acts as a catalyst in the process of hemoglobin production. Many other minerals —manganese, zinc, fluorine, boron, aluminum, vanadium— occur in plants and animals; however, deficiency of any of these in man seems improbable because they are widely distributed in nature and man's requirements of them are minute.

CALCIUM

A warning was given by Hobart Hare[25] that too often the use of calcium means the introducing of an agent about which the physician knows little into a body about which he knows less. Those of us who knew and esteemed this great teacher sense what he meant by this bit of pleasantry. We all felt he knew everything—he certainly knew his Voltaire. Some

two hundred years ago, in not so pleasant a vein, Voltaire[26] put it: "Doctors are men who cram drugs of which they know little into bodies of which they know less, for diseases of which they know nothing at all." But—we are learning! The doctor does not know all, but he does know more than the patient—and to that extent he can guide and help him.

Cantarow[27] noted that the diet of the Finns averages 2.6 gm. of calcium oxide daily—owing chiefly to their large milk consumption; whereas the average daily American diet contains only 0.45 gm.—insufficient for calcium needs—owing to the low intake of milk and dairy products. Children require relatively more calcium. In general a high calcium intake is needed during the growth period; also during pregnancy and lactation, because of the drain on the mother's calcium reserves. Experimental work shows that calcium is absorbed in the small intestine. Mother's milk contains more lactose than cow's milk; this is a factor in the higher degree of utilization of calcium from mother's milk. Lactose promotes a calcium absorption not promoted by other sugars; this is attributed to the acid condition within the intestines due to the lactose. "D" is essential to normal calcium metabolism. In contrast to the other vitamins "D" has an extremely limited distribution in foods; in fact, the only substances in which "D" exists in amounts sufficiently to be of practical importance are egg-yolk, halibut and codliver oils and, to much lesser degree, the cream of cow's milk. Administration of parathyroid extract results in increased elimination of calcium, through the urine.

The calcium intake in China has been estimated by Adolph[28] as less than 0.45 gm., daily. In some nutrition clinics bone meal and limestone have been used to supplement the calcium supply. The suggestion has been made to add a calcium salt to the almost universally used soybean sauce.

Nine cases of cataract are reported by Weekers[29]—seven of which followed thyroidectomy, evidently with inclusion of the parathyroids. Two cases followed idiopathic tetany. Weekers believes that, following parathyroidectomy, cataract may be prevented by giving "D" and calcium. He further

believes that this treatment may check early changes in the lens.

IRON

Inorganic iron is much more widely distributed throughout animal and vegetable tissues than is generally realized; in fact, Heath and Patek[20] say that iron is known to be present in most, if not all, cells—both plant and animal. There is no definite evidence that loss of iron by the body can be produced by limiting the iron intake. Although 12-15 mgm. per diem is usually considered optimal intake, diets containing less than this will maintain the iron balance. There is a marked difference between the metabolism of iron and that of such elements as nitrogen or calcium. The amount of iron excreted in the urine is negligible whereas a loss of nitrogen or calcium may be produced very easily by restricting their intake. Hahn estimates that 57 percent of the iron in the body is in the hemoglobin, 20 percent in storage depots—liver, spleen and marrow—16 percent in various enzymes and 7 percent in muscle. Iron is readily stored in the liver, but it would seem that it is not utilized in the absence of a trace of copper. However, the possibility of a copper deficiency in man is exceedingly remote.

Just because lesions of the oral mucosa may be due to a B Complex deficiency it seems to Darby[21] that we are not justified in assuming that all such lesions reveal a vitamin deficiency. It is claimed that changes in the lingual papillae are specifically due to niacin deficiency; and it has been implied that fissures at the angle of the mouth are pathognomonic of a "B_2" deficiency. Darby quotes Waldenström's[22] clinical report that the administration of *iron*, without any other therapy, resulted in the healing of fissures at the corner of the mouth, sores on the tongue and pains and spasm in the throat. It is possible that patients may have taken a better diet during the period of iron therapy and that the iron may have not been solely responsible for the results observed. However, Darby's own experience leads him to agree that lack of iron alone can account for atrophic lingual papillae,

glossitis and fissures at the angle of the mouth. Many patients with these lesions failed to respond to adequate dosage of B Complex; many gave diet histories which seemed adequate, and these were substantiated by laboratory tests on body fluids. Tests were carried out, using radioactive iron. If an adult absorbed more than 5 per cent of radioactive iron it was assumed that iron deficiency existed. The cases reviewed show that angular fissures and superficial glossitis may accompany a chronic iron deficiency of mild degree. Certain of these cases responded to iron therapy alone; and others responded to iron therapy after failing to yield to prolonged B Complex treatment. It would appear that iron deficiency is a common cause of these oral lesions.

IODINE

As is customary in other neoplastic diseases, Seidlin, Marinelli and Oshry[32] have first attacked adenocarcinoma of the thyroid by surgery, radiation, or both. They mention that metastases usually have received radiation—which is only palliative. They recommend treating these metastases by "internal radiation" with radioactive iodine. The earliest study of this treatment was reported by Hamilton[34] in two cases of carcinoma in 1940. In 1942 Hamilton reported two more cases[35] in which tracer doses of radioactive iodine had been given prior to the removal of carcinomatous thyroid. It has been found that thiouracil has the same systemic effects in cancerous as in simple hyperthyroidism. The authors report three years' observation of radioactive iodine therapy in a case of metastatic thyroid carcinoma. All demonstrable metastases were found to have picked up the radioactive iodine. This patient had a malignant thyroid adenoma removed in 1923. Fifteen years later classic symptoms of hyperthyroidism developed; also severe lower back pain. A pulsating tumor was removed; it proved to be metastatic thyroid adenocarcinoma. In the next two years the hyperthyroidism increased and X-ray showed new metastases in the lungs, femur, rib, ileum and skull. External radiation proved ineffectual and a tracer dose of radioactive iodine was given. Iodine retention was shown in all lesions. There

was no evidence of residual thyroid tissue in the neck. Radioactive iodine was given orally for five months; definite, lasting clinical improvement followed. Later, additional doses were given with resultant disappearance of pain, increase in weight and progressively less evidence of hyperthyroidism. X-ray evidence pointed to arrest, if not regression, of the disease. There were no untoward effects of this therapy.

Thiouracil has recently been widely used in hyperthyroidism. It is given before operation on the thyroid as well as to those in whom operation is contraindicated. For preoperative treatment it is given until the major symptoms have subsided and the basal metabolic rate is normal. During the third and second weeks preceding operation iodine is given with the thiouracil; and during the final week before operation iodine is given alone. In editorials[26], [27] it was noted that sulfonamides and thiourea exert a depressing influence on thyroid activity and hence on the basal metabolic rate. Thiouracil showed certain advantages over thiourea in bringing about these results. It is not without toxic effects; the statistics in several thousand cases showed granulocytopenia in 2.5 percent, leukopenia in 4.4 percent, drug fever and dermatitis. If a significant drop occurs in the white cell count the drug should be stopped at once. Blood transfusion may be advisable. Thiouracil and its derivatives inhibit the formation of the thyroid hormone. Whether complete cure will be possible is not yet known; this will probably depend on the development of less toxic derivatives.

FLUORINE

The prevention of dental caries by adding fluorine to water supplies begins to appear promising, from a survey by Martin[28]. Little fluorine is needed in order to avail consumers of this preventive; and any danger of toxicosis is avoided by limiting the concentration to one in a million. The people of Colorado Springs, where rampant dental caries is almost unknown, have been using water of more than twice this concentration (2.6 per million) for over sixty years with no ill effects. It has been estimated that the addition of needed fluorine to water supplies would cost 5c a person per year.

"A"

For his investigations of the effect of an "A" deficiency upon the bony structure of the labyrinth and secondarily upon the vestibular and cochlear nerves, Mellanby[9] used litters of puppies from seven to ten weeks old. He fed them a diet complete in all respects except for its "A" content; the period of experimentation ran from 5 to 10 months. Bony overgrowth nearly filled some internal auditory meatuses, and thickened the periosteal bone of the capsule especially on the brain aspect; the modioli showed new bony growth. Serous labyrinthitis developed in the cochleae in varying degrees; where bone overgrowth and nerve degeneration were advanced there was always serous labyrinthitis, apparently ensuing upon these changes. The degeneration of the nerve structures brought about complete disappearance of the cochlear nerve, the cells of the spiral ganglion and their central and peripheral branches. To a lesser degree the vestibular division of the VIII Nerve was also affected. The posterior roots of the spinal cord as well as other cranial nerves, such as the optic and the trigeminal, showed similar degenerative changes.

B COMPLEX

The first to associate nerve deafness with a lack of vitamins, especially the B Complex, was Grant Selfridge. It seems to us that the present concept could be best expressed as follows: It appears that nerve deafness can be caused by such a lack; that the deafness is not relieved by giving the vitamins—but that tinnitus has been lessened. To add further to this study Johansen[10] gave the B Complex to 43 patients, most of whom suffered with nerve deafness—some with otosclerosis. None heard better; but in 4 out of 16 patients tinnitus disappeared. If only the patient would consult the otologist sooner, it is reasonable to suppose that vitamin therapy might also help the hearing defect. In one case, seen early, with a rather acute loss of cochlear function, the hearing became normal after "B_1" was given.

"B"

An interesting study of the effect of "B_1" on the distance of the near point was made by Sysi[41] in Helsinki. The subjects examined were not selected for age or refractive conditions; some were hyperopic, some emmetropic, some myopic; "B_1" was given intravenously. Change in the near point took place in about 83 percent—more often in hyperopes than in emmetropes and myopes. In hyperopes larger doses exerted the effect more rapidly; in emmetropes and hyperopes the approach of the near point was found greatest when large doses were given. In a small number the near point was farther away after the injection. The return to the original distance takes place gradually, generally in about three days. Subjects over 60 years of age often manifest a strong response. Considerable accommodative power after 60 years can be detected fairly often. There was no indication in these subjects of any noteworthy lack of "B_1". By way of explanation of this response to "B_1" Sysi offers two possible assumptions—the strengthening of the ciliary muscle, or increase in the elasticity of the lens.

NICOTINIC ACID

The value of nicotinic acid in the case of a healthy man who developed a state of mental confusion after an automobile accident is reported by Lehmann[42]. There was no hint of an avitaminosis. For six weeks he was restless, uncooperative and violent. He was finally moved from a general hospital to an institution receiving only mental and nervous patients. Hyperpyrexia treatment in the second week resulted in some clearing of the sensorium, but his Korsakoff syndrome remained unimproved. In the fourth week Lehmann resorted to the use of nicotinic acid, giving 250 mg. by mouth, and 50 mg. intravenously, daily for seven days. By the end of the week the patient became the composed, cheerful and intelligent man he had been before the accident; two months later he resumed his occupation as an accountant. Lehmann finds an explanation of this dramatic effect in the vasodilator action of nicotinic acid, and considers that the relative hyperemia of the brain improved the cerebral metabolism.

Mental disturbance, varying from drowsiness to pronounced lethargy, was the outstanding feature in a series of patients suffering from acute pellagra; Cleckley, Sydenstricker, and Geeslin[28] observed remarkable improvement following the use of nicotinic acid. Nineteen patients were so treated, and of these six are described in detail. Three patients were delirious, and one showed agitated depression. Not all cases showed the usual signs of pellagra; but in four the tongue showed typical glossitis, and two suffered from vaginitis. Nicotine acid in the form of sodium nicotinate was given daily, 100 cc. to 300 cc. intravenously, for periods ranging from 3 to 12 days. The beneficial effect of the vitamin was seen in some of these stuporose patients in as short a time as four, five or six hours. The authors are convinced that this therapy was life-saving; and that further, in many such individuals who have succumbed, their pellagra presented cerebral symptoms without other recognizable marks of the avitaminosis.

FOLIC ACID

We should be more familiar with folic acid, which is assuming importance in eye, ear, nose and throat practice. Occasional leukopenia and granulocytopenia incidental to drug administration have been the bane of all clinicians for twenty years or more. Daft and Sebrell[29] induced such blood impoverishment in rats by giving sulfaguanidine or sulfasuxidine; the total leukocyte count fell to 4,000 or less. Folic acid solutions were given by mouth for four consecutive days, with an average increase of 14,400 leukocytes, and an average increase of granulocytes from 1 to 39 percent.

By combining one of the sulfa compounds (succinylsulfathiazole) with food in an amount of 1 percent of the total, Endicott, Daft and Ott[30] were able to study the effect of this drug on the bloodforming systems of rats. Leukopenia and granulocytopenia develop regularly on this regimen; bone marrow becomes hypercellular, and there is lymphoid exhaustion of the spleen. The response to folic acid is rapid and spectacular; the spleen shows lymphoid regeneration, and

the bone marrow becomes greatly enriched in its cellular content even to the degree of overcompensation. Their earlier investigations on the effectiveness of folic acid in cases of leukopenia in rats encouraged Watson, Sebrell, McKelvey and Daft[45] to use it in the human subject. They preface their report by emphasizing that in laboratory animals the blood abnormalities had been brought on by combining a sulfonamide with food. In their series of 17 clinical cases 10 were instances of refractory anemia and toxic leukopenia, and of these only 2 showed sulfonamide therapy as a factor; in 5 there was a history of insecticide exposure and of dilantin administration; there was 1 case of Hodgkin's disease in which Roentgen rays had been used for treatment, and in the remaining 2 causative factors were not known. The results of folic acid treatment were nil. The second group comprised 7 cases of leukopenia as a sequel to Roentgen ray therapy, employed in 1 case for polycythemia vera and in 6 for carcinoma of the cervix uteri. In this group, as in the former, folic acid did not prove of value; to be sure, there was a rather consistent increase of leukocytes after its use but there was doubt whether this could or could not be credited definitely to the folic acid. The authors undertook this investigation as a "mandatory experiment," and plead for further study in leukopenia cases, especially when radiation treatment presents itself as a probable weighty factor.

Many case studies have been made to evaluate the action of folic acid in pernicious and related macrocytic anemias. Vilter, Vilter and Spies[46] investigated its effects in 28 cases of pernicious anemia, sprue and macrocytic anemia for a period of one year. Almost uniformly the blood picture changed to normal. Many had received liver extract before this investigation; but liver therapy was discontinued for the purpose of this study. The point of major interest was that 4 of the pernicious anemia cases manifested combined system disease, symptoms of which were weakness in the legs, numbness and tingling in the hands and feet and other evidence of peripheral neuritis. The daily dosage of folic acid (10 to 50 mg.) was increased very greatly (100 to 500 mg. daily),

but, even so, failed to correct the neuritis. In two cases, liver extract, 50 U.S.P. units, was injected intramuscularly daily and brought about rapid improvement. In a third case, the signs of neuritis appeared and disappeared independent of the folic acid therapy. The inference is that some unknown factor or factors are present in liver and are essential for maintaining the integrity of the central and pheripheral nervous systems in cases of pernicious anemia; folic acid, therefore, while strikingly effective for the blood abnormalities, does not replace liver in cases complicated by nervous disorders.

In association with Lopez and Menendez of Havana, Spies, Minnich and Koch[47] transferred the study of folic acid in sprue from the Southern States to Cuba, where new clinical evidence confirmed the value of synthetic folic acid. They selected three patients for investigation, and found that persons suffering from macrocyctic anemia of sprue in relapse are greatly benefited by folic acid given by mouth in the amount of 10 mg. daily.

Further investigation of folic acid and its synthetic derivatives led Spies, Lopez, Milanes and Aramburu[48] to appraise the value of synthetic pteroyldiglutamyl—glutamic acid. They treated a 55-year old patient suffering from tropical sprue with this compound, giving 10 mg. daily by intramuscular injection for eight days. His red cells increased from 1.32 million to 2.43 million in the short period of five days, and his hemoglobin from 43 to 58 percent.

The fundamental defect in sprue appears to be impaired absorption of various substances from the gastro-intestinal tract, as a result of which a deficiency of a number of factors may be brought about. Characteristic findings in the disease are glossitis, diarrhoea with increase of fat in the stools, loss of weight, pigmentation of the skin, macrocyctic anemia, moderate leukopenia, impairment of absorption as shown by a flat oral glucose curve and "A" tolerance curve, a very low serum carotene, and a characteristic gastro-intestinal pattern on X-ray examination. These patients were treated by Darby, Jones and Johnson[49] with synthetic L. casei factor

("folic acid") given intramuscularly in a dosage of 15 mg. daily for 18, 19 and 15 days respectively. In all cases the soreness of the tongue ceased after four days; and in two the diarrhoea was relieved by the eighth and twelfth days; but the fat content of the stools did not become normal. There was in all cases a renewed sense of well-being; the response in terms of red cells, white cells and platelets was "complete". In one patient, a woman whose treatment was undertaken six weeks after childbirth and whose store of iron was presumed to be low, a macrocytic anemia was recognized eight weeks after the treatment ended; in her case a relapse was to be expected. All the cardinal manifestations of sprue which are amenable to liver therapy undergo remission as a result of the intramuscular injection of synthetic L. casei factor. The question of dosage is not yet finally answered; 15 mg. in daily injections is probably more than the smallest amount needed for the best effects; in fact a maintenance dose may be of the order of 0.1 to 0.2 mg. daily and a curative dose as little as 1.0 mg. daily.

"C"

Studies of wound healing have been made through the ages. In recent years this process has been found to depend to a great extent on the presence of "C". Experiments on animals have given satisfactory evidence but clinical observations have been incomplete. It is a rarity for man to exhibit a deficiency of a single vitamin. The human organism cannot synthesize "C". The tensile strength is directly proportionate to the "C" content of the scar tissue tested. An opportunity was afforded Wolfer, Farmer, Carroll and Manshardt[50] to study the effect on wound healing of a prolonged "C" deficiency in 9 otherwise normal, healthy young males. In addition 5 healthy males were available as controls. Each subject was a medical student who volunteered to be deprived of "C" for a period of 7 months; chemical analysis of meals showed the "C" content to be either very low or nil. The deficiency was established and maintained throughout the 7 months. At the end of this time an incision was made in the thigh through the skin and fascia, and the course of wound

healing studied by removal of biopsy specimens at intervals of 4 to 14 days. The conclusions were that the lack of "C" resulted in a 50 percent diminution in the tensile strength of the healing wounds. Such a depletion delays the development of tensile strength from 3 to 5 days. When conditions for healing are poor, such as the presence of undue wound tension or diminution in blood supply, there is gross failure of primary wound healing. The observation of wound infection suggests the need of "C" for resistance to infection by favoring primary wound healing. These human subjects exhibit a lack of collagen and reticulum in their healing wounds.

"D"

For a long time it had been noted that cod-liver oil was of benefit in tuberculosis. In 1884, 74 cases of lupus were reported by Emery as being cured by the enormous dosage of 1,000 gm. of cod-liver oil daily. The next advance in the treatment of lupus was the use of heliotherapy and of concentrated ultraviolet light applied locally by Finsen in 1897. The next step was the isolation of "D" from cod-liver oil and the preparation of "D_2" by exposing ergosterol to ultraviolet light, thus effecting the isolation of pure calciferol ("D_2") in 1931. Feenie[51] reports the treatment of 150 cases of lupus vulgaris. The dose of calciferol was 100,000 units daily, and this was increased to 150,000 units in some cases. Local treatment also was given in 87 cases, and general light baths in 14 cases. The success attained would have taken twice or three times as long before the introduction of the "D_2" therapy. Almost all the cases became independent of the general light treatment. For the first 9 months "D_2" appears to be increasingly efficacious. The proportion of resistant cases was virtually the same as it had been before the advent of "D_2". There were signs of toxicity in 8 cases, but in the 2 cases in which arterial calcification occurred it was possible to resume treatment with "D_2" after an interval. Throughout, routine X-rays of the kidneys and arteries were made—for signs of calcification.

Although we are deeply interested in the present food

problem of Great Britain, it is not for us to discuss whether centralized control is better, or whether the comment of the Conservatives rings true—that the laborer is saying to the Government: "You promised us less work and more pay. Well, we are doing our part—we work less." However, either a world shortage or their present form of government has brought about a lack of food which the German submarine failed to accomplish. At our request a highly intelligent Englishman has written us the human side[22] as of today: "Our lacks vary, but are never serious in the sense of mass starvation or the threat of epidemic. Just now our greatest lack is milk—one and one half pints a week per person. Until recently we could buy American dried skimmed milk occasionally; it costs us 'points' but we paid them gladly. We can no longer stomach "full cream" dried milk; it makes us sick—for we haven't even seen cream since 1939. Each shortage, in itself, is trivial. We get twenty cents worth of meat a week.* If that were our only privation we would not notice it. However, this is the latest of a series, which has progressively increased for seven years. As to vitamins, we do not lack those we really need. My family takes a B Complex addition to food, with frequent doses of black currant juice; and we grow a small amount of fruit and vegetables. Our bread is better than it was before the War when it was atrociously 'refined' into worthlessness. Bread, however, is rationed. Those of us who are either young or not too poor can manage to get enough of a monotonous diet to avoid hunger and undue fatigue."

One wonders whether the British will manage to secure enough essential nutrients during the present crisis. Is it even possible that their privations may be fraught with some benefit? Compare their plight with that of us Americans. We eat too much. Of all the food consumed in the United States half of it is unnecessary; and of that half a large proportion contributes to the physical delinquency of the eater. The quantity is excessive; the quality, deplorable—notably in

*Official prices, Oct., 1947: beef (thin flank) 11c per lb.; (ribs) 27c per lb.; (imported frozen ribs) 22c per lb.; mutton 30c per lb. Strangely, in spite of vastly greater supply, we are paying about 400% more.

refined carbohydrates and sweets. Early in the War the official pronouncement was that it was fortunate for the people of the British Isles that they were to be denied sugar for a sufficiently long period to derive great benefit from its absence.

Today we as a nation are faced with a paradox. The President has recently called a special session of Congress to consider not only the mounting costs of food at home but also the crying need for food abroad. Yet the health of the average American would be improved if he ate 1000 calories less a day. If, in addition, the criminal waste of food in hotels, restaurants and homes could be stopped, what a contribution to the World's food shortage!

BIBLIOGRAPHY

1. JONES, ISAAC H.: Vitamins and the Eye, Ear, Nose and Throat. THE LARYNGOSCOPE, 51:615-625, July, 1941.

2. COVELL, WALTER P.: Vitamins and the Ear. THE LARYNGOSCOPE, 51: 683-691, July, 1941.

3. JONES, ISAAC H.: Vitamin Therapy Today. THE LARYNGOSCOPE, 52:811-814, October, 1942.

4. COVELL, WALTER P.: Pathologic Changes in the Peripheral Auditory Mechanism due to Avitaminoses. THE LARYNGOSCOPE, 50:632-647, 1941.

5. MICKEY, KARL B.: Book. Health from the Ground Up. International Harvester Co., Chicago, 1946.

6. PRICE, WESTON A. Book: Nutrition and Physical Degeneration. Paul B. Hoeber, New York, 1939.

7. MAYNARD, LEONARD A.: A Chemist's View of Nutrition. *Science*, April 18, 1947, 399-403.

8. EDITORIAL—The Teaching of Nutrition—II. *Journal A. M. A.*, 134:1, 23-34, May 3, 1947.

9. EDITORIAL—The Teaching of Nutrition—III. *Journal A. M. A.*, 134:2, 146-147, May 10, 1947.

10. MACY, ICIE G., PH. D.; and WILLIAMS, HAROLD H., PH. D.: Book. Hidden Hunger. The Jaques Cattell Press, Lancaster, Pennsylvania, 1945.

11. WESTERMAN, BEULAH and HALL, GERTRUDE: Improving the Nutritive Value of Flour. *Journal of Nutrition*, 33:301, March 10, 1947.

12. BILL HENRY, Columnist: *The Los Angeles Times*, November 6, 1947.

13. WILLIAMS, ROBERT: Synthetic Vitamins and Human Nutrition. *Industrial and Engineering Chemistry*, 39:464-466, April, 1947.

14. BISKIND, M. S.; and WILLIAMS, R. R.: Incidence of Avitaminotic Lesions and Effects of Therapy. *Journal of Digestive Diseases*, 121-130, April, 1947.

15. KNAPP, A. A.: The Eye as a Guide to Latent Nutritional Deficiency Diseases. *Bulletin N. Y. Acad. Med.*, 22:217-222, April, 1946.

16. WHITBOURNE, DAHLIA: Nutritional Retrobulbar Neuritis in Children in Jamaica. *Amer. Jour. Ophth.*, 130:169-171, February, 1947.

17. NARDINI, JOHN E.: Vitamin-Deficiency Diseases in Allied Prisoners of the Japanese. *U. S. Naval Med. Bull.*, 47:272-278, March-April, 1947.

18. CLECKLEY, H. M.; SYDENSTRICKER, V. P.; and GEESLIN, L. E:. Nicotinic Acid in the Treatment of Atypical Psychotic States, Associated with Malnutrition. *Jour. A. M. A.*, 112:2107-2110, May 27, 1939.

19. MCCOY, C. M.; MAYNARD, L. A.; SPERLING, G.; and OSGOOD, H. S.: *Jour. Nutrition*, 45-60, January, 1941.

20. RUSCH, HAROLD P., M. D., and BAUMAN, CARL A., PH. D.: Nutritional Aspects of the Cancer Problem. *Nutrition Reviews*, 353-355, December, 1946.

21. ELMAN, ROBERT: The Practical Use of Amino Acids in Protein Nutrition. *Jour. A. M. A.*, 128:659-664, June 30, 1945.

22. STARE, FREDERICK J., and THORN, GEORGE W.: Protein Nutrition in Problems of Medical Interest. *Jour. A. M. A.*, 127:1120-1127, April 28, 1945.

23. GLUCK, REGINA and WILSON, JAMES L.: Fever Produced by the Oral Administration of Amino Acids. *Amer. Jour. Dis. Children*, 71:601, June, 1946.

24. HARDING, T. SWANN: *Minerals in Nutrition.* Medical Record, 134-138, March, 1947.

25. HARE, HOBART A., Foreword of Book by CANTAROW, ABRAHAM: Calcium Metabolism and Calcium Therapy. Lea & Febiger, Philadelphia, 1933.

26. VOLTAIRE: Book. Sir Arthur Helps Friends in Counsel, second series, Chapter 10. Published by Routledge, 1907.

27. CANTAROW, ABRAHAM: Calcium Metabolism and Calcium Therapy. Lea & Febiger, Philadelphia, 1933.

28. ADOLPH, WILLIAM H.: Nutrition Research in China. *Jour. of Amer. Dietetic Ass'n*, 22:964-970, November, 1946.

29. WEEKERS, R.: Cataract Due to Low Blood Calcium and Its Medical Treatment. Ophthalmologica, 107:257, May-June 1944. Abstracted by F. H. Adler, in *Arch. of Ophthl.*, 792-793, June, 1947.

30. HEATH, CLARK W., and PATEK, ARTHUR J., JR.: The Anemia of Iron Deficiency. XVI, 1937, 267-350.

31. DARBY, WILLIAM J.: The Oral Manifestation of Iron Deficiency. *Jour. A. M. A.*, 130:830-835, March 30, 1946.

32. WALDENSTROM, J.: Iron and Epithelium. Some Clinical Observations. *Acta med. Scandinav.*, supp. 90:380, 1938.

33. SEIDLIN, S. M., M. D.; MARINELLI, L. D., M. A.; and OSHRY ELEANOR, B. S: Radioactive Iodine Therapy—Effect on Functioning Metastases of Adenocarcinoma of the Thyroid. *Journal A. M. A.*, 132:838-847, Dec. 7, 1946.

34. HAMILTON, J. G.; SOLEY, M. H.; and EICHORN, K. B.: Deposition of Radioactive Iodine in Human Thyroid Tissue. Univ. California Publ., *Pharmacol.* 1:339-368, 1940.

35. HAMILTON, J. G.: The Use of Radioactive Tracers in Biology and Medicine. *Radiology*, 39:541-572, Nov., 1942.

36. EDITORIAL. Thiourea and Thiouracil in Treatment of Thyrotoxicosis. *Jour. A. M. A.*, Sept. 16, 1944, 172.

EDITORIAL. Hyperthyroidism. Jour. A. M. A., Feb. 22, 1947. 544-545.

38. MARTIN, NOEL D.: Dental Caries; Public Health Aspects of Control by Fluorine. *Med. Jour. Australia*, 486-490, April 19, 1947.

39. MELLANBY, EDWARD: The Experimental Production of Deafness in Young Animals by Diet. THE LARYNGOSCOPE, 49:1090-1118, Nov., 1939.

40. JOHANSEN, HENRIK: Treatment of Hardness of Hearing and Tinnitus with Vitamin "B". *Acta Oto-laryngologica*, 34:177-181, March to June, 1946.

41. SYSI, R.: Investigations on the Effect of Vitamin B_1 on the Accommodation of Man. Acta Ophth. Suppl. 1946, 128. (Reported in *Quart. Review of Ophth.*, Vol. 2, No. 4, Dec., 1946.)

42. LEHMANN, H.: Post-Traumatic Confusional State Treated with Massive Doses of Nicotinic Acid. *Can. Med. Assoc. Jour.*, 51:558-560, Dec., 1947.

43. DAFT, FLOYD S.; and SEBRELL, W. H.: The Successful Treatment of Granulocytopenia and Leukopenia in Rats with Crystalline Folic Acid. *Public Health Reports*, 58:1542-1545, October 15, 1943.

44. ENDICOTT, K. M.; DAFT, FLOYD S.; and OTT, MAURINE: The Bone Marrow in "Folic Acid" Deficiency and its Response to Crystalline Lactobacillus Casei Factor ("Folic Acid"). *Arch. of Path.*, 40:364-372, November-December, 1945.

45. WATSON, C. J.; SEBRELL, W. H.; MCKELVEY, J. L.; and DAFT, F. S.: Possible Effectiveness of the L. Casei Factor ("Folic Acid") Concentrates on Refractory Anemia and Leukopenia, with Particular Reference to Leukopenia Following Radiation Therapy. *Amer. Jour. of Med. Sc.*, 210:463-470, October, 1945.

46. VILTER, CARL F.; VILTER, RICHARD W.; and SPIES, TOM D.: The Treatment of Pernicious and Related Anemias with Synthetic Folic Acid. 1. Observation on the Maintenance of a Normal Hematologic Status and the Occurrence of Combined System Disease at the End of One Year. *Jour. of Lab. and Clin. Medicine*, 32:262-273, March, 1947.

47. SPIES, TOM D.; LOPEZ, GUILLERMO GARCIA; MENENDEZ, JOSE ARISTILES; MINNICH, VIRGINIA; and KOCH, MARY B.: The Effects of Folic Acid on Sprue. *Southern Medical Journal*, 30:30-32, Jan., 1946.

48. SPIES, TOM D.; LOPEZ, GUILLERMO GARCIA; MILANES, FERNANDO and ARAMBURU, THOMAS: Synthetic Folic Acid the Effectiveness of Conjugated Form in the Treatment of Tropical Sprue, Addisonian Pernicious Anemia and Nutritional Macrocytic Anemia. *Jour. A. M. A.*, 134:18-20, May 3, 1947.

49. DARBY, WILLIAM J.; JONES, EDGAR and JOHNSON, HOWARD C.: Effect of Synthetic Lactobacillus Casei Factor in Treatment of Sprue. *Jour. A. M. A.*, 130:780-786, March 23, 1936.

50. WOLFER, JOHN A.; FARMER, CHESTER J.; CARROLL, M. A.; WALTER, W.; MANSHARDT, DONALD O.: An Experimental Study in Wound Healing in Vitamin C Depleted Human Subjects. *Surgery Gynecology and Obstetrics*, 84:1-15, January, 1947.

51. FEENIE, P. J.: Calciferol in Tuberculosis. Review of 150 Cases of Lupus Vulgaris. *The Lancet*, XII:438, April 5, 1946.

52. Personal Communication. GROUP CAPTAIN G. STRUAN-MARSHALL, (ret.) Edinburgh, Scotland.

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THE RECRUITMENT PHENOMENON AND DIFFERENCE LIMEN IN HEARING AND VIBRATION SENSE.*

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Summary: In nerve deafness the normal relation between the physical intensity of the sound and the loudness perceived by the subject is disturbed in a characteristic manner. As the sound intensity is increased, loudness increases more rapidly than is normal. This is known as the recruitment phenomenon. When it appears, the changes in sound intensity that can just be perceived are smaller than normal; that is, the difference limen for loudness is diminished. This effect was directly confirmed.

Since it is easier to measure the difference limen than to use the previous method of measuring recruitment, this difference limen was used to show that recruitment appears not only in hearing but also in the vibration sense in general. It has, therefore, a general physiological significance. Exactly the same phenomena are encountered in nerve deafness as are found in vibration sense during freezing, or when the sense organs are fatigued by vibrations of large amplitude; furthermore, it could be shown that the measurement of the difference limen reveals a commencing nerve deafness much earlier than does the hearing threshold itself.

1. Introduction.

Since the formulation of the Weber-Fechner law, the way in which loudness increases as the strength of the sound stimulus increases has come to possess a general psychological and physiological interest. This interest has grown since it was shown, especially by E. P. Fowler,[1] that the relation

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existing between the strength of the stimulus and the perceived loudness is modified by nerve deafness in a different manner than it is by conduction deafness. Clinical problems were thus brought immediately into the orbit of these investigations. The effect found in nerve deafness is that the loudness perceived by the patient grows more rapidly than is normal when the sound intensity is increased. Fowler calls this the recruitment phenomenon.

Hitherto, recruitment has been demonstrated only with hearing. The question arises, however, whether it is really absent in other senses or has not been demonstrated because of the lack of suitable and sufficiently simple methods of measurement. Certainly the measurement of the effect in the case of hearing, performed as it has been in the past by loudness comparisons, cannot be regarded as simple. The equations of loudness, which have been required in measurements of the recruitment phenomenon, have always suffered in practice from the difficulty in discriminating between the attribute of loudness and other effects, such as timbre or transients. The accuracy of measurement is therefore always poor. Even a telephone engineer usually needs a few days before he can perform comparisons with an accuracy of 2.0 db.

The recognition of the difference limen for variation in sound intensity is comparatively simple. This difference limen may be measured at the hearing threshold itself; that is to say, one may measure the amount by which the amplitude must be increased in order to make a certainly inaudible sound become audible. In this case, as observations on nearly 100 patients have shown, a few minutes are sufficient to enable the subject to become proficient; furthermore, measurements of difference limens have the advantage over judgments of equal loudness that the measurements may be confined to quite a narrow frequency range.

Every change of loudness implies a certain change in sound intensity; but when there is recruitment, a given change of loudness requires a much smaller change in sound intensity in the diseased than in the healthy ear. This has been shown

by comparisons of the loudness perceived by a defective ear and a healthy ear in the same subject. When the magnitude of the loudness change is diminished, this difference between the defective and healthy ear persists. When the smallest just-perceptible changes of loudness are reached, it is to be expected that the requisite increment in sound intensity, that is, the difference limen, will be diminished when the recruitment phenomenon is present.[2] It seems, therefore, that the difference limen at the threshold of hearing is especially well suited to the examination of patients suffering from nerve deafness.

The arrangement of equipment used in such measurements has already been described[3] and is shown in Fig. 1. The telephone receiver producing the test tone is connected to a potentiometer, the setting of which is automatically and continuously varied at constant rate by an electric motor. The movement of the potentiometer is so arranged that, when the control button is pressed, the loudness increases, and when it is released, reversing the motor, the loudness diminishes. The patient presses the button until the inaudible sound becomes just audible, and he then releases the button until the sound has again become inaudible. The extent of the variation in potentiometer setting is recorded on a drum and gives a measure of the difference limen at the absolute threshold of hearing. By gradually changing the frequency and simultaneously rotating the drum, we obtain an "audiogram" of the difference limen at different frequencies over the entire audible region. Fig. 2 shows an audiogram of this kind in a case of nerve deafness (a), and for comparison in a case of conduction deafness (b).

For most of the vibration measurements a potentiometer with steps of 0.4 db. was used with the speed of potentiometer movement adjusted to 1 db. per second. In another potentiometer these two values were halved.

2. The Recruitment Phenomenon in the Vibration Sense.

From the purely physiological standpoint, it has always

been found that a close relation exists between the sense of hearing and the sense of vibration; accordingly, it became of interest to examine vibration sense for possible recruitment effects.

By fatiguing a normal ear one can very easily produce deafness which shows the recruitment phenomenon very nicely.[4] It, therefore, appeared worthwhile to see whether the recruitment phenomenon with vibration sense could not also be produced artificially, especially since eliciting the effect artificially makes it much easier to demonstrate small changes.

To do this, the oscillator (see Fig. 1) was disconnected from the motor shaft, so that the drum rotated alone. The frequency of the oscillator was kept constant at 100 c.p.s. for most experiments. The telephone receiver was replaced by a

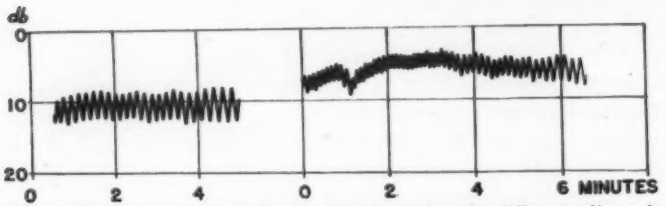

Fig. 1. Arrangement of equipment for measuring the difference limen by means of intensity changes at the hearing threshold.

vibrating system which moved an ebonite button of about 2 mm. diameter. The vibrations of this ebonite button were transverse, that is, parallel to the surface of the skin, so that the stimulus remained as superficial as possible. The impedance of the vibrating system was great enough to eliminate changes in the amplitude of movement when the button was in contact with the skin surface. The variations in vibration threshold at the fingertip and the corresponding difference limens were recorded as a function of time. Next, the fingertip was sprayed with ethyl chloride until anesthetized, and the measurements continued. With this method, only occasional changes in the difference limen were produced. The lack of any clear effect may be ascribed to the spreading of the vibration over the entire finger, even with such a small

vibrating button. The values measured really belong to the skin surface around the anesthetized area.

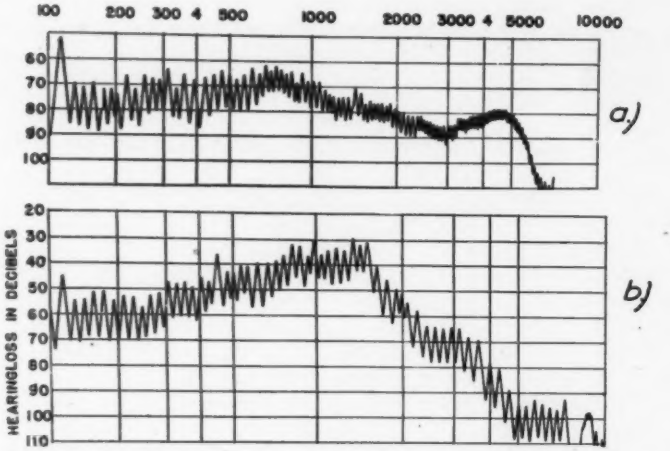

Fig. 2a. Diminishing difference limen with increasing frequency in nerve deafness.

Fig. 2b. Constant difference limen in conduction deafness.

Fig. 3 shows the reduction of the vibration as it passes along the index finger, using a frequency of 100 c.p.s. and a

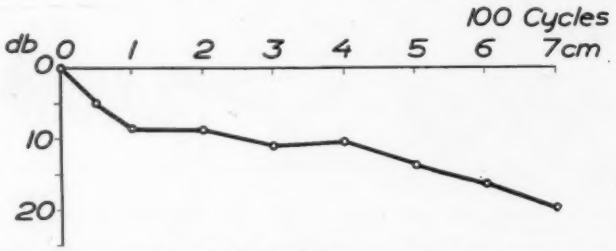

Fig. 3. Reduction of vibration spreading along the index finger when the vibrating system is applied at the tip. Frequency, 100 c.p.s.

vibration applicator of 2 mm. diameter. The measurements here were made with a very light dynamic pickup. With higher frequencies there is much less spreading, but then the

adaptation of the vibration sense becomes so great that continuous measurements cannot be made over a sufficient period of time.

The tip of the nose seemed more suitable for these measurements because of the gradual transition from soft skin surface to cartilage and bone.

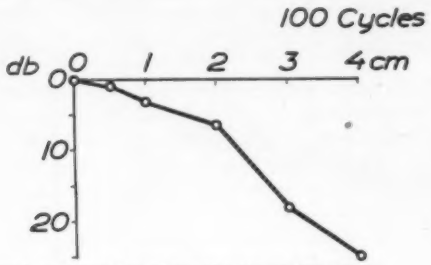

Fig. 4. Spread of vibration along the tip of the nose.

Fig. 4. shows the reduction in the amplitude of vibration of a frequency of 100 c.p.s. in relation to the distance from the place of application on the tip of the nose. With ethyl chloride freezing, it was here possible to produce, in many though not all cases, variations in the difference limen similar to those found in hearing when the auditory sense is fatigued.

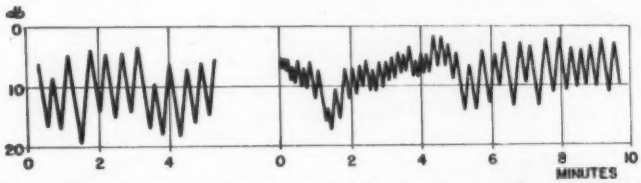

Fig. 5. Recruitment phenomenon with vibration sense after freezing with ethyl chloride.

Fig. 5 shows, at the left, the difference limen which occurs normally; and at the right, its magnitude after freezing.

Extremely strong tingling, corresponding to tinnitus, arose when the finger was frozen, and this predominated over the

sensation from the vibrational stimulus. On the tip of the nose, however, this effect did not appear, nor was the pulse a disturbing factor at this site. It was found advantageous to protect all of the nose except the tip by a shield so that all excess ethyl chloride would flow away. At any place where a drop remained, there was no evaporation and, therefore, no freezing. Even then it was very difficult to arrange the anesthesia so that the measurements recorded really had reference to the actual spot being examined.

It appeared desirable, therefore, to use the same stimulus for the anesthesia as was used for measurement. If this were done, the anesthetizing and the test stimulus had necessarily to be applied to the same site. This was accomplished by the

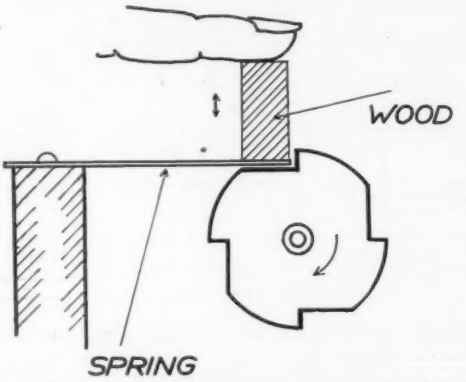

Fig. 6. Rotating toothed wheel for producing fatigue by vibrations of high amplitude.

use of vibrations of large amplitude which fatigued the local area and the underlying deep sensibility. The fatiguing was done by means of a ratchet wheel, shown in Fig. 6.

Vibrations of 100 c.p.s. and an approximate amplitude of 3 mm. were applied in this way for one-half to one minute. The fatigue produced was sufficient to show the reduction in the difference limen and the subsequent recovery very well. The effects are quite analogous to those with hearing. On the

finger the anesthesia cannot be carried very far because the heat generated by internal friction is too painful for the fatiguing to be continued for long. On the tip of the nose or on the arm, however, such limiting difficulties were not found.

Fig. 7 shows measurements of the threshold for the vibration sense, before and after fatigue produced by vibrations of 100 c.p.s. Such results could be repeated readily. The recovery rate varies widely with different persons.

The absolute value of the threshold was raised by only about 2 to 5 db. as Fig. 7 shows.

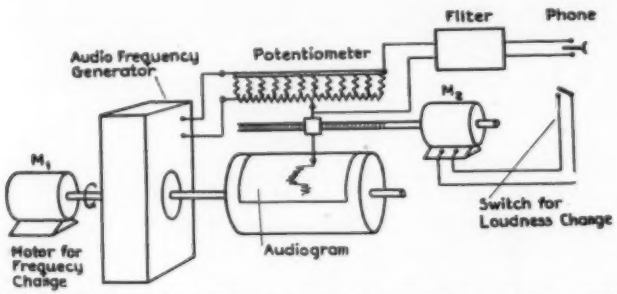

Fig. 7. Reduction of the difference limen for vibration sense after fatigue.

3. Diminution of the Difference Limen.

In order to investigate the related effect at the threshold of hearing, a series of sounds of constant frequency were presented, increasing in amplitude about 1 db. every two minutes. The amplitude was kept constant at each level between the successive increases. The first sound was set at a value just under the hearing threshold. The manner in which the successive sound signals in the series are perceived by the normal ear is represented in Fig. 8, which shows the temporal effects at each separate stage. The top line *a* represents the subthreshold stage where no sound whatever can be heard. When the sound intensity has been increased by about 1 db., the signal can just be perceived at irregular moments. The perception occurs in quite short phases of equal magni-

tude represented by the separate zones *b* of Fig. 8. In the next stage there is an increase in the rate at which the brief phases of sensitivity appear, but the perceived strength of the sound remains unaltered. The actual physical intensity of the sound has, of course, been increased by 1 db. from the preceding stage. A further increase in the intensity of the sound produces a continuous excitation interrupted only for short intervals *d*. Finally, with a further increase, the sound is heard as completely continuous, although somewhat fluctuating. This is shown at *e*, where it will be seen that the perceived phases vary in magnitude.

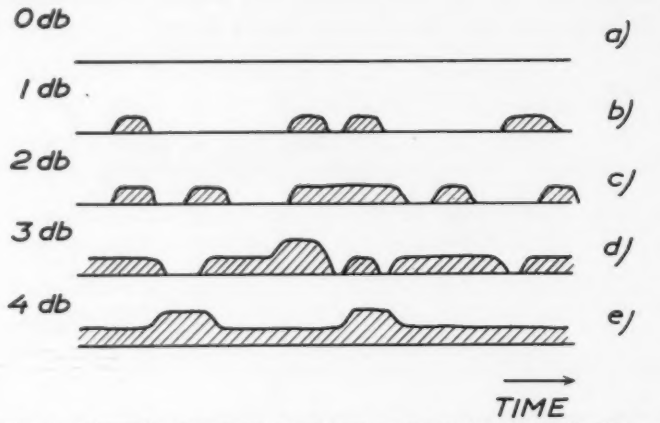

Fig. 8. Schematic representation of how sounds of different intensity are heard in the neighborhood of the hearing threshold.

The ratio of the pressure amplitude of sound at which continuous excitation first appears, to the maximum pressure amplitude which cannot be heard at all, is called the difference limen at the absolute threshold for the frequency in question. It is measured by the range of the excursions up and down in the audiogram of Fig. 2. With vibration sense, the succession of effects described above may be followed even better than with hearing. The separate phases in the changing sensitivity of the skin corresponding to *b*, Fig. 8, arise from separate regions of the skin surface, with the

result that they can very well be felt as separate. The vibrational amplitude can also be readily estimated, owing to the integration into a unified broad surface experience of the sensory impacts that arise at diverse points.

If, using the audiometer described, the vibration threshold of any selected region of the arm is measured with the tip of a needle vibrating at a frequency of 100 c.p.s., large variations of the difference limen appear as the needle point slips over the surface of the skin, a movement it makes unavoidably by itself. Usually the vibrating system can simply be held in the hand because sufficiently large variations become evident in time. The difference limen is thus found to change by a ratio of more than 1:5, as Fig. 9 shows.

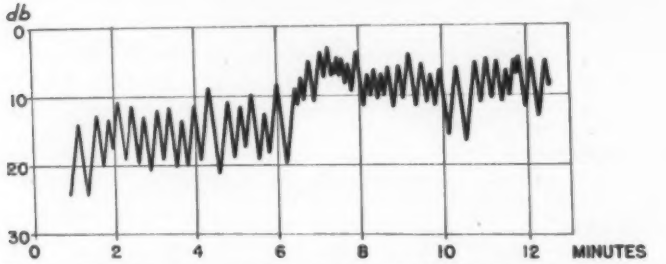

Fig. 9. Variation of threshold and of the difference limen for vibration sense when a vibrating needle moves over the skin surface.

By this method, too, it may be observed quite clearly in regions where the difference limen is large that, when the vibrational amplitude is increased, the corresponding increase in the sensation develops from widely scattered small excitations which arise in the zone surrounding the point of application of the primary stimulus. The density of these distributed impulses becomes greater toward the center of the area until they finally fuse into a continuous excitation at the point of stimulus. The summation of these quite irregular and small impulses, scattered in direction and distance, gives rise to a sensation which appears to be widely spread.

In places where the difference limen is small, however, the

foregoing summation effect is absent. There is then only one sensation which appears almost instantaneously. This difference between a diffuse and a point-localized excitation may be clearly distinguished. In the case of point-localized sensation, moreover, if the amplitude of the vibrations is increased at the rate of 0.5 db./sec., usually only two or three impulses are felt separately before continuous excitation sets in. With diffuse excitation under the same conditions, on the other hand, the discrete impulses are much more frequent and appear at longer time intervals.

When the audiometer is adjusted to increase the amplitude of vibration at the rate of 0.5 db./sec., the initial vibratory experience may be either a diffuse, widespread sensation, or

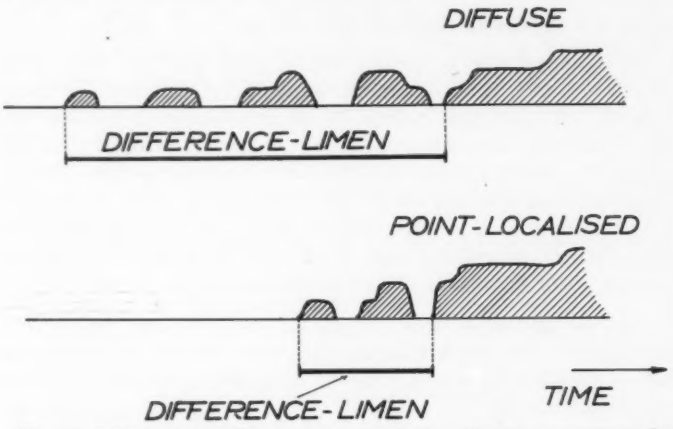

Fig. 10. Effects during the commencement of a vibratory experience for the diffuse and the point-localized types of sensation. Time is measured horizontally; perceived magnitude of stimulus, vertically. The amplitude of vibration is increasing at the same rate in each case.

a point-localized sensation, the temporal course of which is shown schematically in Fig. 10. The respective difference limens are also shown by the length of the light horizontal bars.

If, in place of a needle, a surface of 20 mm. diameter is used to apply the vibrational energy to the skin, the vibratory

experience is always diffuse in character and has a considerably larger difference limen; furthermore, it was found that in those regions of the skin where both the pressure-sensitive points and the vibration-sensitive points lie close together, it was seldom possible to localize a transition zone between areas having a larger or smaller difference limen. Where the separation of the vibration-sensitive points was large, however, as in the upper arm, this offered no difficulty.

From this observation it may be concluded that, when only one or a few vibration-sensitive points are excited, the difference limen, as shown in Figs. 8 and 10, is small; but in those regions of the skin where there are many vibration-sensitive points simultaneously excited, the difference limen is much larger because of the summation of a number of separate, irregular points of excitation.

This conception is in agreement with the fact that anesthesia of the skin by freezing or fatigue causes a large number of vibration points to become insensitive. The difference limen then diminishes because of the decrease in the effective number and density of sensitive points. Under anesthesia it may be observed very readily that, even with the application of a vibrating stimulus 10 mm. in diameter, only the point-localized type of experience arises and there is no fusion of scattered points of excitation.

By analogy with these effects in the vibratory sense it is conceivable that with hearing, too, the number of functional nerve endings is diminished in diseased conditions of the nerve, so that the difference limen is correspondingly reduced. Similarly, since the stimulated area of the basilar membrane is large, with low frequencies, and becomes progressively smaller as the frequency increases, it is to be expected that the decrease of difference limen as a result of disease will be less marked for low frequencies. More than 100 audiograms have been made which show this effect (see Fig. 2b).

Finally, it was found that the threshold for the vibratory sense at which continuous excitation appears was almost identical for both the diffuse and the point-localized types of

sensation; moreover, anesthesia causes more marked changes in the difference limen than in the threshold itself. It follows, therefore, that the difference limen should be a better measure of the commencement of aural disease than the hearing threshold itself. Almost all audiograms showed that as the frequency was gradually increased, the difference limen was the first to diminish, while the reduction of the absolute hearing threshold appeared only later at still higher frequencies. Fig. 2a shows this quite clearly.

These investigations have been concerned only with the phenomenological aspects of the effect. The nerve mechanism producing them is at present unknown.

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REFERENCES.

1. Fowler, E. P.: *Arch. Otolaryngol.*, 8:151, 1928; 24:731, 1930.
Fowler, E. P.: The Laryngoscope, 47:289, 1937.
Steinberg, J. C., and Gardner, M. B.: *Jour. Acoust. Soc. Am.*, 9:11, 1937.
Steinberg, J. C.: The Laryngoscope, 47:511, 1937.
Brinnitzer, W.: *Monatsschr. Ohrenheilk.*, 69:1301, 1935.
De Mare, Goran: *Acta oto-laryngol.*, Suppl. XXXI, 1939.
Huizing, H. C.: *Audiometerie*, 1941.
Huizing, H. C.: *Neder. Tijds. hr. v. Geneesk.*, 85:4529, 1941.
De Briune-Altes, J. C.: The Symptom of Regression in Different Kinds of Deafness, 1946.
Bekesy, G. v.: *Physik. Ztschr.*, 30:721, Fig. 1, 1929.
2. Brinnitzer, W., and De Mare, Goran, have given consideration to the difference limen in nerve deafness.
3. Published in *Acta oto-laryngol.*
4. Bekesy, G. v.: *Physik. Ztschr.*, 30:721, Fig. 1, 1929.

REPRESENTATION OF THE COCHLEA IN THE CEREBRAL CORTEX.*

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The process of hearing requires the functioning of a mechanism for analysis of sound into its component frequencies. This mechanism must be able to operate throughout a wide range of sound intensities. Most investigators now believe that the mechanism in which these functions are carried out is the organ of Corti and related structures. The early correlation of high-tone deafness in patients with atrophy of the organ of Corti in the basal turn is perhaps the most solid evidence for this belief. A number of investigators have obtained confirmatory evidence from animal experimentation using condition reflexes, Pryer reflexes, cochlear potentials, etc., as an index for hearing. In general, all these experiments led to the conclusion that high tones are localized in the basal coil and successively lower tones are arranged in order of frequency toward the apex. These experiments depended either on demonstrating maximum electrical activity for a particular tone at a given region of the cochlea or the destruction of localized regions of the organ of Corti, demonstrating the resulting impairment of cochlear function.

Tonal Localization in the Cochlea: In 1942, Dr. John Bordley and I[1] performed some experiments on cats in which we made localized lesions in the organ of Corti by drilling away the bony capsule over the spiral ligament and depressing a small area of this structure without opening into the endo-lymphatic spaces. Lesions in the basal coil caused high tone loss, and lesions toward the apex caused low tone loss (Fig. 1). We also found in these experiments that the number of decibels of impairment depended on the length of the lesion. This was true for either one long lesion or a number of small lesions separated by normal

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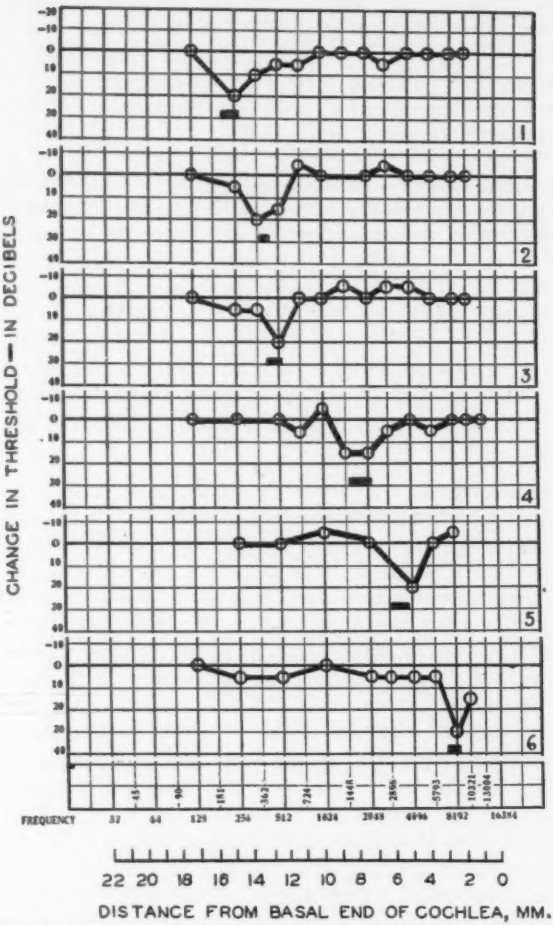

Figure 1. Changes in thresholds of cochlear response to pure tones following localized lesions in various parts of the cochlea.

The short black line in each audiogram indicates the position and length of the lesion with reference to the scale at the bottom of the figure.

organ of Corti. We concluded that at threshold there is sharp localization of tones along the organ of Corti, but that

as the intensity is raised, there is spread of response to adjacent areas of the organ of Corti. Ingenious experiments by Galambos and Davis[2] on response in single fibers of the eighth nerve have added greatly to our understanding of these findings and support the conclusion that there is spread of response with increase in intensity.

Tonal Localization in the Cerebral Cortex of the Cat: If the analysis of sound is accomplished in the organ of Corti, the pattern of response that is set up must be maintained intact through the higher pathways, with their several synapses, to the auditory cortex. Up to a few years ago most clinical and experimental evidence failed to support this theory. Clinical evidence in man was inconclusive because the bilateral representation of hearing made it unlikely that a lesion would selectively effect a part of the hearing range, since such a lesion would have to be bilateral and symmetrical.

Previous attempts to show localized response in the cortex experimentally were unsuccessful mainly due to the difficulty of obtaining a short, pure tone without a click. Dr. Clinton Woolsey and I[3] tried to demonstrate cerebral tonal localization by stimulating the organ of Corti electrically through holes drilled over the spiral ligament. We found there was too much electrical spread in the perilymphatic fluids; however, we did find it possible to stimulate small bundles of nerve fibers in various parts of the cochlea after removing the bony capsule and the organ of Corti and placing small electrodes in contact with the exposed nerve fibers in the osseous spiral lamina. The nerve fibers were stimulated with short condenser discharges. The exposed temporal lobes of the cortex were systematically explored with a wick electrode and the responses recorded by means of a cathode-ray oscillograph. In these experiments, when near threshold intensities of stimulation were used, stimulation of the nerve fibers in the basal end of the cochlea caused a narrow band of responses to appear in the anterior superior part of the auditory area. Stimulation of apical coil fibers caused responses in the posterior superior part. Stimulation of fibers in intermediate parts of the cochlea resulted in potentials in

intermediate parts of the auditory area. (See Fig. 2.) We found in some experiments, in which stronger stimulation or lighter anesthesia was used, that potentials also occurred in ventral parts of the area. Stimulation of cochlear nerve fibers in the basal turn under these conditions caused responses in the posterior inferior part as well as the anterior superior; and apical turn stimulation caused responses in the anterior inferior as well as the posterior superior. The ventral areas were usually more or less separated from the dorsal

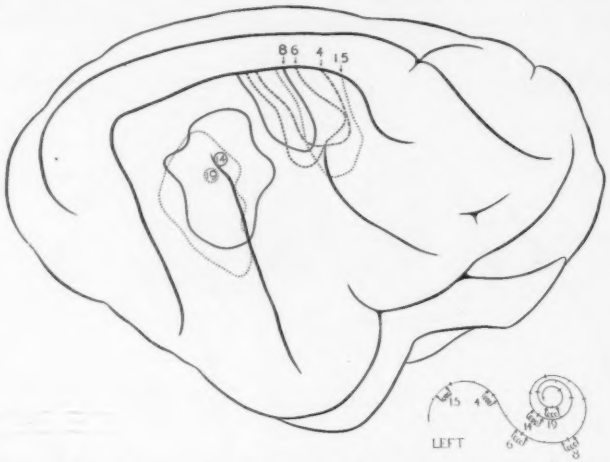

Figure 2. Diagram of lateral aspect of the cerebral cortex of the cat, showing focal areas of cortical response for stimulation of nerve fibers in a number of points in the cochlea (1.5, 4, 6, 8, 14, and 19 mm. from the basal end).

areas by zones of minimum response (Fig. 3). We concluded from these experiments that there is point-to-point projection of the cochlear nerve to the auditory area of the cerebral cortex and that there are dual auditory areas in each hemisphere arranged in inverted order with respect to the various parts of the cochlea. We also found in these experiments that stimulation of one cochlea caused symmetrical and equal response in both cerebral hemispheres, demonstrating bilateral representation of hearing. Almost coincidentally with our report, Ades and Culler[4] obtained other evidence for

cerebral tonal localization. They found that lesions in the medial geniculate body caused lack of response in localized regions of the auditory area to click stimulation. They had previously shown that such lesions caused differential hearing loss as shown by conditioned reflexes.

Projection Pathways from the Medial Geniculate Body: Waller[5] had previously shown that lesions in various parts of the auditory area of the cortex caused chromatolysis in localized regions of the medial geniculate body. On re-examining his data in the light of our findings, it would appear that there are two projection systems in the cat from the

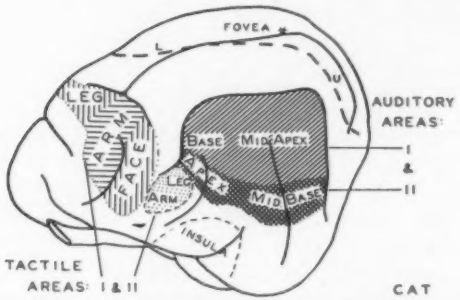

Figure 3. Diagram of the lateral aspect of the cerebral cortex of the cat, showing the position of the Auditory areas I and II and their internal organization with relation to the parts of the cochlea. The relationship to Tactile areas I and II are also shown.

medial geniculate body to the cortex. Fibers from the small cell part of the nucleus apparently go to the first auditory area while fibers from the large cell part of the medial geniculate body go to the second auditory area.

Cochlear Representation in the Cortex of the Monkey: In an attempt to extend the studies on localization of the auditory cortex to forms closer to man, we repeated our experiments on the Macac monkey[6]. The auditory area of the monkey is largely buried within the sylvian fissure so that exploration required removal of either the dorsal or ventral bank. Since the auditory area is located partly in the upper and partly in the lower bank, no single experiment could be

complete; and the conclusions represent the synthesis of results of a number of experiments. In some animals, the dorsal bank on one side was explored and the ventral bank on the other. Preliminary exploration with click stimulation revealed that the auditory area occupies approximately the posterior half of the lower bank of the sylvian fissure ending at about the level of the posterior end of the insula. Some responses were also obtained from click stimulation on the inner part of the upper bank. Stimulation of nerve fibers in the basal coil of the cochlea caused response in the posterior medial part of the area. Stimulation of apical turn fibers caused response in the anterior lateral part of the area (Fig. 4). In the most medial portion of the area, there was definite crossing of the basal and apical turn areas. This,

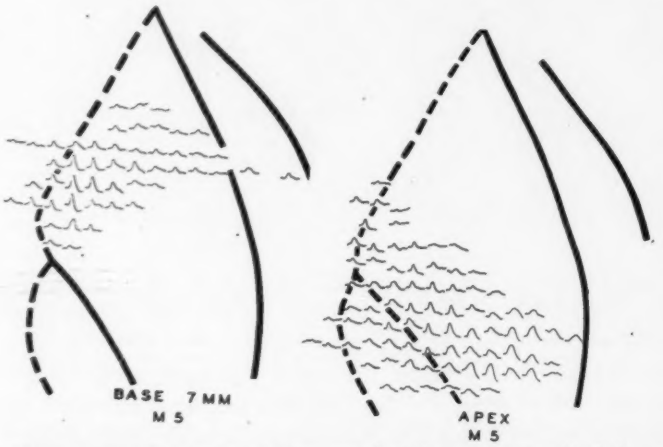

Figure 4. Response in the auditory cortex of the monkey to stimulation of nerve fibers in the basal and apical turns of the cochlea.

undoubtedly, represents the second response area for the monkey. These responses were found to extend also onto the upper bank. The relationship of the second response area to the second somatic area as found in the cat is maintained in the monkey (Fig. 5).

Effect of Cochlear Lesions on Auditory Response in the Cortex: Although the experiments described above demonstrated that the pattern of nerve fibers in the cochlea is maintained intact as far as the auditory cortex, we did not have direct evidence that response to pure tones is localized in the cortex. Previous attempts to demonstrate tonal localization in the cortex had failed because of the difficulty of obtaining short bursts of pure tones without producing a click. We attempted to circumvent this difficulty by studying the effect of impairment of hearing to a particular part of the sound spectrum on the auditory response area in the cortex as shown by click stimulation[7]. The procedure followed was to map the total auditory cortex by means of click stimulation; then produce a high or low tone loss, shown by cochlear

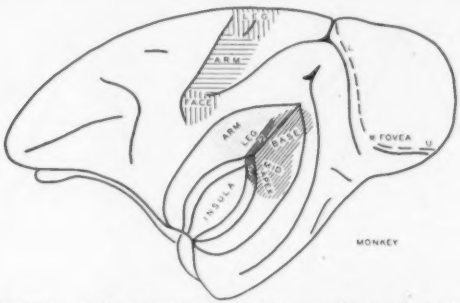

Figure 5. Diagram of the lateral aspect of the cerebral cortex of the monkey. The sylvian fissure is shown as if spread open, showing the position of Auditory areas I and II, and their relation to Tactile area II for arm and leg (dotted area).

potentials, by depressing the spiral ligament in the appropriate part of the cochlea. The auditory area was then remapped to click. A lesion in the basal coil, producing marked high tone loss, caused disappearance of potentials in the anterior superior and posterior inferior parts of the auditory area (Fig. 6). Conversely, lesions in the apical coil of the cochlea causing low tone impairment were followed by disappearance of potentials in the posterior superior and anterior inferior part of the auditory area. The areas in which potentials disappeared are consistent with what we know about the projection pattern of the cochlear nerve. In all of these

experiments, there was clear evidence of the duality of auditory representation in each hemisphere.

In some of our experiments, only slight impairment of hearing was obtained, and in these experiments there was no loss of response anywhere in the cortex; however, there was increased threshold in the regions characteristic for the impaired tones. This can be interpreted as additional evidence for spread of the response in the cochlea with increase in the intensity of sound.

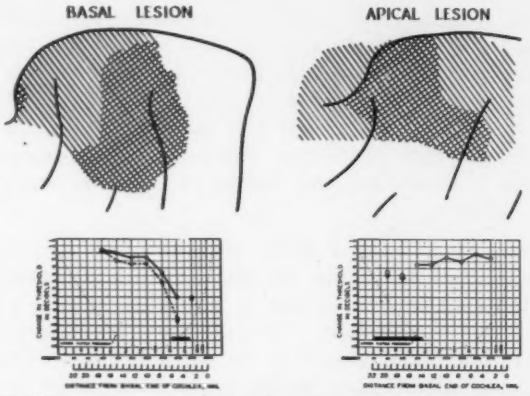

Figure 6. Audiograms and diagrams showing effect of localized lesions in the cochlea of the cat on thresholds of cochlear potentials to pure tones, and the corresponding change in the area of response to click stimulation in the auditory cortex.

The cross-hatched area in each case represents the region in which responses continued to occur following the cochlear lesion.

DISCUSSION.

Since these experiments were first described, Licklider and Kryter[8] and McCulloch[9] have been able to obtain localized response in the cortex to short bursts of pure tones. This was accomplished by the use of a mechanism for gradually increasing the intensity of sound without producing a click. Their results are in good agreement with ours. Our results have also been confirmed by Tunturi in the dog. Tunturi[10] claims to have found a third auditory area in the dog in the region of the junction of the anterior ecto-sylvian and

coronal gyri. He found little evidence of tonal localization in this region. Dr. Woolsey and I have looked for auditory response in this area in both cats and dogs and have failed to find any; however, we were not using the same anesthetic which may account for the difference. Allen[11] has obtained some confirmatory evidence for the presence of Tunturi's area in studying the effect of ablation of the three known auditory areas on condition reflexes. He found it necessary to ablate areas one and two in order to get any loss of conditioned differential auditory response. Even with areas one and two completely ablated, the response was quickly re-acquired; however, if area three were also ablated, the response was abolished for a considerable period. Ades[12] has also been able to demonstrate a secondary or association area in the posterior ecto-sylvian gyrus by the use of strichninization of the first area. McCulloch[9] also found in using the strichnine technic that there are point-to-point interconnecting pathways from one auditory cortex to the other through the corpus callosum.

In conclusion, we now know that there is a spatial frequency localization in the cochlea and that the pattern set up in the cochlea by a sound is projected to dual auditory areas in each hemisphere. The dual areas are arranged in inverted order with respect to the parts of the cochlea, i. e., the auditory nerve fiber in the basal turn projects to the anterior part of area one and the posterior part of area two. Two additional auditory areas have been demonstrated. One is clearly an association area, while the function of the other area is still uncertain.

REFERENCES.

1. WALZL, E. M. and BORDLEY, J. E.: The effect of small lesions of the organ of Corti on cochlear potentials. *Amer. J. Physiol.*, 1942, *135*: 351-360.

2. GALAMBOS, R. and DAVIS, H.: The response of single auditory nerve fibers to acoustic stimulation. *J. Neurophysiol.*, 1943, *6*: 39-57.

3. WOOLSEY, C. N. and WALZL, E. M.: Topical projection of nerve fibers from local regions of the cochlea to the cerebral cortex of the cat. *Bull. Johns Hopkins Hosp.*, 1942, *71*: 315-344.

4. ADES, H. W. and CULLER, E. A.: Effect of lesions in the medial geniculate bodies upon hearing in the cat. *Amer. J. Physiol.*, 1939, *125*: 15-23.

5. WALLER, W. H.: Thalamic degeneration induced by temporal lesions in the cat. *J. Anat.*, 1939-40, *74*: 528-536.

6. WALZL, E. M. and WOOLSEY, C. N.: Cortical auditory areas of the monkey as determined by electrical excitation of nerve fibers in osseous spiral lamina and by click stimulation. *Fed. Proc. Amer. Soc. Exp. Biol.*, 1943, *2*: 52.

7. WALZL, E. M. and WOOLSEY, C. N.: Effect of cochlear lesions on click responses in the auditory cortex of the cat. *Bull. Johns Hopkins Hosp.*, 1946, *79*: 309-319.

8. LICKLIDER, J. C. and KRYTER, K. D.: Frequency localization in the auditory cortex of the monkey. *Fed. Proc. Amer. Soc. Exp. Biol.*, 1942, *1*: 51.

9. MCCULLOCH, W. S.: The functional organization of the cerebral cortex. *Physiol. Rev.*, 1944, *24*: 390-407.

10. TUNTURI, A. R.: Further afferent connections to the acoustic cortex of the dog. *Amer. J. Physiol.*, 1945, *144*: 389-394.

11. ALLEN, W. F.:Effect of destroying 3 localized cerebral cortical areas for sound of correct conditioned differential responses of the dog foreleg. *Amer. J. Physiol.*, 1945, *144*: 415-428.

12. ADES, H. W.: A secondary acoustic area in the cerebral cortex of the cat. *J. Neurophysiol.*, 1943, *6*: 59-63.

LARYNGOCELE.*

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Under the following heads I am going to make some general observations in connection with laryngocele: definition, history, incidence, etiology, pathology, symptoms, diagnosis and treatment; and then I will speak briefly of two cases of our own.

Definition: Laryngocele has been defined as an anomalous air sac connected with the larynx. Perhaps that definition is not quite complete, because it should state that it is a hernia of the larynx, which is implied in the name itself. It is most commonly from the laryngeal ventricle, and it must be lined with mucosa to be a true laryngocele. It is not a tumor, but must be considered under the diagnosis of tumors because benign tumors and malignant tumors figure importantly in the differential diagnosis.

History: It is very interesting that Larrey, the famous physician and surgeon connected with Napoleon's armies, observed in Egypt these gaseous tumors or air sac tumors in priests who chanted the verses of the Koran from the minarets. Also, he noticed it in drill sergeants and he mentions two instances in his writings, but it was in 1867 that Virchow first definitely described laryngocele as a clinical entity called ventricular laryngocele.

Marshall Taylor,[1] in his paper read before the American Laryngological Association in 1944, gave a complete review of the history and also the more recent literature of laryngocele. There are many papers in the literature, most of them reporting, as I am today, one or two cases but discussing the subject in a general way at the same time.

Incidence: It was formerly said in practically all papers

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written about laryngocele that it was a rare condition. The word "rare" has been used over and over again. We are coming to believe that it is not so rare as was previously thought. It is a condition very often overlooked and still being overlooked, occurring either alone or in association with other conditions.

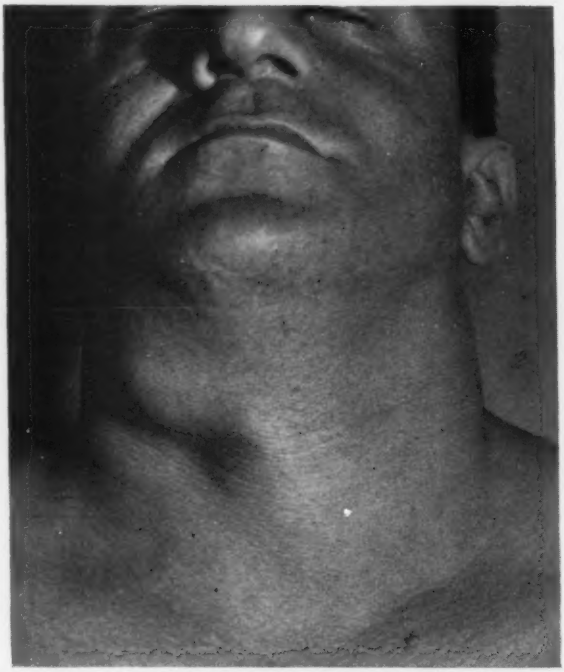

Fig. 1. Appearance of neck before operation for right external laryngocele (Case 2).

Schall[2] reported an interesting case in which he found laryngocele associated with cancer of the larynx. Leborgne,[3] of Montevideo, a radiologist who has written a beautifully illustrated book on the study of cancer of the larynx from the radiological viewpoint, feels that the incidence of associated laryngocele and cancer of the larynx may be as high

as 10 or 15 per cent. Leborgne was one of the pioneers in the use of the planigraphic method in the study of the larynx and because of that, he has been able to pick up so many cases.

Etiology: Atavism is always mentioned in discussing the etiology of laryngocele, and Taylor gave considerable space to this subject in his paper. The relation with the air sacs of some kinds of monkeys is evidence in favor of the atavistic factor in etiology. The laryngocele seems to be an elongated ventricular appendix. Despite these atavistic features there are other etiologic factors which are also unquestionably important.

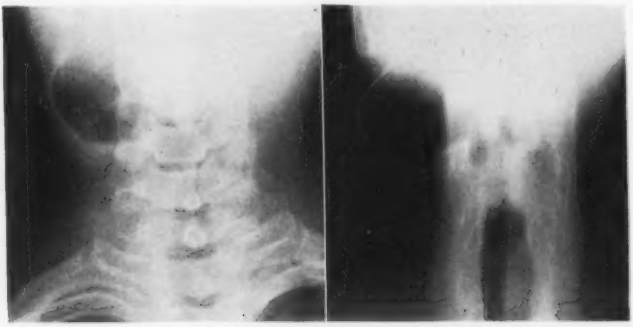

Fig. 2. Roentgenographic appearance of laryngocele, ordinary film to the left and planigraphic film to the right. Note that the planigraphic film shows the communication between the external laryngocele and the internal or ventricular portion (Case 2).

Anything that increases the intralaryngeal pressure — coughing, singing, blowing a horn — all of those etiologic factors have unquestionably played a part in some of the cases; for example, Taylor's patient was a singer.

There seems to be a check valve action at the narrow bottleneck of the laryngocele. It has been proven quite well by X-ray and particularly planigraphic studies, that air gets into the sac and pressure builds up, because air doesn't escape so readily from the sac.

The association with other conditions is very important. Boies[4] wrote an interesting report of two cases of papilloma of the larynx which he reported under that heading, "Unusual Cases of Papilloma," and which was published in THE LARYNGOSCOPE. He called attention in this article to the fact that the laryngocele seemed to be a secondary effect of the obstruction caused by the papilloma. One of the cases that I am reporting today is also a case of associated papilloma and laryngocele.

Fig. 3. External laryngocele removed by external operation (Case 2).

Pathology: I have already said a true laryngocele must be lined with mucous membrane. There are air collections in the region of the larynx which are not lined with mucous membrane, but they are not called laryngoceles. Virchow made the distinction between *external* and *internal* laryngocele, the internal being the one inside the thyroid cartilage and the external being the one that is outside the thyrohyoid membrane so it could be seen as a bulge in the neck. One of our cases is of this type.

A third group has been mentioned by some writers, a mixed group. Leborgne in his excellent discussion of the subject points out that this differentiation between the internal, external and mixed is really not very sound, that the internal

laryngocele is a clinical entity, but that the external would be better called an exteriorized form because it is really internal in its nature. In the first place, it herniated out through the thyrohyoid membrane and thus has become an exteriorized lesion. The mixed is really not a necessary classification, because any external laryngocele is really mixed, so the suggestion made by Leborgne is very sound.

When we speak of internal laryngocele or one that has become externalized, associated conditions should be considered under pathology, too, and perhaps the effect produced by papilloma and carcinoma, syphilis and several other conditions in connection with the formation and perpetuation of the laryngocele. Another thing that should be mentioned under pathology is infection, because in a number of cases the laryngocele has become infected and sometimes this has been a complicating factor in treatment.

Symptoms: What are the symptoms? Effect on the voice is the most important, that is, hoarseness, but most of these patients notice some dyspnea as well. Certainly if the sac has increased beyond a certain point in size, if it has become external, there is an external bulge. Another sign is the hissing sound, the sound of air passing back into the larynx if you make pressure on the external bulge. That sign was present in the patient that we operated upon and are reporting (Case 2). This bulge is generally increased on coughing and, of course, you get a tympanitic note on percussion. The laryngeal appearance usually is first a thickening of the ventricular band on the affected side. Sometimes the laryngocele appears as an isolated tumor mass and may even be pedunculated.

Diagnosis is made on the basis of the symptoms, the laryngeal appearances, the external appearance, if it is one of the cases in which the laryngocele is herniated out through the thyrohyoid membrane, but more on the X-ray and particularly the planigraphic X-ray studies. They are the means of making a definite diagnosis and giving a graphic representation of the size and relations of the lesion.

Leborgne states that in every case planigraphy will demon-

strate, if the section is taken in the proper place, the external part of the laryngocele and the communication between the ventricle and sac. Lipiodol has been injected in some cases to prove the diagnosis, and that apparently didn't entail any serious consequences; but it is not necessary to inject lipiodol, and certainly I prefer not to use it.

Finally, *treatment:* if it is associated with conditions such as papilloma, carcinoma or syphilis, of course, they call for treatment, obviously. As far as laryngocele itself is concerned, in many of the cases of internal laryngocele, biting out some of the sac wall endoscopically is doubtless indicated. In Boies' case that was done. In one of the cases reported by Alonso,[5] of Uruguay, external surgery was done to excise an internal laryngocele. In that case, Alonso first did a tracheotomy and then by external approach excised the internal laryngocele. In the externalized laryngoceles, I think unquestionably surgical excision is indicated, as done by Taylor in his case, and as we did in one of the cases herein reported.

CASE REPORTS.

Case 1: A male, age 22. Hoarseness for many years, pain and soreness occasionally, due, he thought, to colds or excessive use of the voice. Twelve or 14 years previously, he stated he had had several operations for "cysts of the larynx." We weren't able to find out just what those operations were, but at one time he had a tracheotomy. The tube was kept in about one month. At the same time he had marked hoarseness and slight dyspnea on exertion. An examination of the larynx showed some bulging, definite reddish nodular tumor tissue occupying the free margin of the right cord. There was a thin-walled, air-filled sac which collapsed during quiet breathing. Externally there is a scar of low collar incision, the site of previous tracheotomy.

The papilloma was removed, the histologic diagnosis confirming the clinical impression, and the interesting thing in this case was that after we cleaned out the papilloma by sev-

eral treatments, the air sac was much reduced in size. Without anything being done directly to it, it became practically unnoticeable by mirror examination and, likewise, was reduced so much by X-ray examination that we didn't think it necessary to do anything else. This man was last seen three years after the papilloma had been treated. At that time, there was no residual papilloma and the laryngocele could not be demonstrated.

Case 2: A man, 35 years of age, who had a swelling on the right side of the neck, hoarseness at times, and especially when he felt the "cell," as he called it, was distended. Two years previously he had noticed a hard swelling on the right side with considerable pain. At that time he was given sulfathiazole and his symptoms were relieved by a few days' administration of sulfathiazole and the spontaneous evacuation of pus through the larynx and the mouth. That was two years previous to the time that he presented himself at our clinic.

We saw the patient first in January, 1944. At that time he had no pain, no evidence of persisting infection, but the sac was blown up on his voluntary effort, and on coughing and some squeezing we could hear the air return into the larynx. We advised surgical extirpation of the laryngocele and that was done. The technique of the operation: We incised the neck on the right side transversely between the hyoid bone and the thyroid cartilage. The platysma fibres were divided and the laryngocele exposed. The laryngocele was then dissected free, retracting the ribbon muscles anteriorly. The sac was closely related with the undersurface of the hyoid bone and the upper edge of the right ala of the thyroid cartilage. It was finally dissected down to a stalk which entered the thyrohyoid membrane almost at the midline. The sac was then ligated and amputated. The cut end was treated with phenol and then inverted with chromic catgut sutures and alloy steel wire.

The operation was done Aug. 31, 1944, and we saw the patient for the last time Oct. 20, at which time the scar was almost invisible, the voice was quite good and X-ray showed

no persisting evidence of laryngocele. Nothing was done with the small internal part of this sac.

REFERENCES.

1. TAYLOR, H. M.: Ventricular Laryngocele. *Tr. Am. Laryngol. Assn.*, 66:114-126, 1944. Also, *Ann. Otol., Rhinol. and Laryngol.*, 53:536, Sept., 1944.

2. SCHALL, LEROY A.: Laryngocele — Associated Cancer of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 53:168, Mar., 1944.

3. LEBORGNE, FELIX: *a.* Estudio Radiológico de Laringocele. *Anales de Oto-R.L. del Uruguay*, p. 239, 1939. *b.* Cáncer de la Laringe. *Estudio Radiologico*, 1943.

4. BOIES, LAWRENCE R.: Papilloma of the Larynx. A Report of Two Unusual Cases. THE LARYNGOSCOPE, Feb., 1943.

5. ALONSO, JUSTO M., and CAUBARRERE, NICOLAS L.: Laringocele. *Anales de Oto-Rhino-Laringologia del Uruguay*, 14 (Parte I):38-44, 1944.

ADDITIONAL REFERENCES.

6. JACKSON, CHEVALIER, and JACKSON, CHEVALIER L.: Diseases and Injuries of the Larynx. Textbook, The Macmillan Co., New York, 1942, p. 358. Also, Plate VIII, No. 10.

7. JACKSON, CHEVALIER, and JACKSON, CHEVALIER L.: Diseases of the Nose, Throat and Ear. Textbook, W. B. Saunders Co., Philadelphia, 1946, p. 589.

8. LINDSAY, J. R.: Laryngocele Ventricularis. *Ann. Otol., Rhinol. and Laryngol.*, 49:661, Sept., 1940.

9. MCLAURIN, J. W.: Laryngocele — Report of a Case. THE LARYNGOSCOPE, 57:616, Sept., 1947.

10. SIMPSON, W. L.: Laryngocele. *Ann. Otol., Rhinol. and Laryngol.*, 47:1054, Dec., 1938.

11. SHAMBAUGH, G. E.: Laryngocele Ventricularis. *Tr. Am. Laryngol. Assn.*, 1914. Also, *Ann. Otol., Rhinol. and Laryngol.*, 24:129, Mar., 1915.

12. LATRAVERSE, VALMORE: *Union Med. du Canada*, 73:890-893, Aug., 1944.

13. OREGGIA, JUAN CARLOS: Los Laringoceles. *Anales de Oto-Rhino-Laringologia del Uruguay*, 1938, fasc. I.

BILATERAL GRANULOMA OF THE LARYNX FOLLOWING ENDOTRACHEAL ANESTHESIA.*

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Granuloma of the larynx as a complication of endotracheal anesthesia is apparently extremely infrequent. For that reason the report of such a case seems justified.

REPORT OF CASE.

A 25-year-old white woman was first seen April 29, 1946, with the complaint of hoarseness of 10 days' duration. Examination revealed nothing abnormal except chronically infected tonsils and injection of the arytenoids. Absolute voice rest was advised, and she was told to return in a week.

She was not seen again until May 11, 1947, when she was referred for tonsillectomy by her family physician. She stated that during the past year she had had frequent upper respiratory infections and had been hoarse at these times but not at any other time. The hoarseness always cleared up promptly and completely under the usual regimen of voice rest. Preoperative examination revealed diseased tonsils but nothing else abnormal; the larynx was entirely negative. Tonsillectomy was performed June 3, 1947, under cyclopropane-ether anesthesia, administered intratracheally, through a Magill No. 35 tube, which was introduced under direct vision, without difficulty. Operation, which lasted 45 minutes, was without incident. The postoperative course was also uneventful, and the patient was discharged June 17, 1947.

She returned July 8, complaining that she had been persistently hoarse since June 20 and that during the last three days pain had radiated from the throat to the ear on each side. Examination at this time revealed bilateral granulomas of the vocal cords. The growth on the right was polypoid and frankly pedunculated; the growth on the left had a somewhat sessile base.

Surgical removal of the granulomas was advised, but the patient was told that it would be better to postpone operation until the growth on the left had also become pedunculated. She was extremely apprehensive, however, because of a recent death in the family from carcinoma of the larynx, and, therefore, both growths were removed at once, under local analgesia, through the anterior commissure laryngoscope, by means of the angular vocal nodular forceps. The cord on the right presented a normal appearance as soon as the growth was removed. The cord on the left presented a raw area over the vocal process.

Hoarseness and pain were completely relieved immediately following operation, but recurred within two weeks, hoarseness was persistent but pain occurred only occasionally. Examination showed that

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the granuloma on the left was re-forming. The patient was now willing to delay operation until the growth had become pedunculated; therefore, operation was deferred until August 22, 1947, when removal was effected without difficulty, by the technique employed at the first operation. The cord seemed entirely normal at the conclusion of the operation.

There has been no recurrence of either symptoms or growths in the three-month period which has elapsed since the second operation. The pathologist reported all the growths as granulomas.

COMMENT.

Granuloma of the larynx as a possible complication of endotracheal anesthesia is not mentioned in most textbooks of anesthesia. Lundy,[1] who states that he has seen "one or two such cases," seems somewhat doubtful that the cause and effect relationship is clearcut and points out that contact granulomas are found in cases in which the endotracheal tube is not used. He admits, however, that occasionally the surface of one or both vocal cords may be traumatized as the tube is passed and that months later, the small blister or hematoma which results may, present as a granuloma.

Only five cases of granuloma of the larynx following endotracheal anesthesia seem to have been reported in the periodical literature to date. The first was reported by Clausen[2] in 1932, the second by Gould[3] in 1935, the third by Cohen[4] in 1938, the fourth by Smiley[5] in 1940, and the fifth by Kearney[6] in 1946. My own case is thus the sixth to be recorded. It is naturally not possible to draw conclusions from six cases, but certain facts of interest and possible significance may be mentioned.

In the reported cases the initial operations were of several different types. Smiley's and Cohen's cases followed operations on the biliary tract, Gould's followed a Wertheim operfor carcinoma of the cervix, Kearney's followed removal of a cyst of the left upper malleolus through a radical antrum approach, Clausen's followed removal of wisdom teeth, and my own followed tonsillectomy. The tubes used were variously described as soft rubber catheters or Magill tubes. In Cohen's and Gould's cases introduction was done blindly and with difficulty. In the remaining cases introduction was under direct vision. In Kearney's case moderate difficulty was ex-

perienced, but Gould stated that in his case introduction was accomplished easily, without trauma, with the patient in the proper position, when the cords were well relaxed. There was no difficulty in my own case. Operation lasted 3 hours and 10 minutes in Gould's case, 95 minutes in Smiley's case, and 85 minutes in Kearney's case, the length of time being explained in each instance by the nature of the procedure or by some operative difficulty. In the other cases, including my own, operation was not unduly prolonged.

In Kearney's case and in my own the larynx had been examined just before operation and was known to be normal, which disposed of the possibility that the granulomas might previously have existed. In Smiley's case it was assumed to be normal, on the ground that there was no preoperative notation that it was not. In the other cases preoperative examination was apparently not done. The growth was bilateral in Kearney's case and my own, and unilateral in the other cases.

In Smiley's case the patient expectorated blood-stained mucous for several hours after operation and became hoarse within ten days; the granuloma was observed on the fifteenth day. Gould's patient began to experience voice failure while she was still in the hospital and was intermittently hoarse for the next three months; she had two attacks of choking, wheezing and cyanosis. Cohen's patient had slight pain in the chest for a day or two after operation; she experienced voice changes and temporary aphonia for ten weeks before she felt something moving up and down in her throat when she spoke. Kearney's patient had a pain in the throat for a week after operation. It recurred five weeks later, associated with hoarseness, and a month later the growth had to be removed as an emergency procedure because of dyspnea and noisy respiration. In Clausen's case the granuloma was found five months after operation, but the symptons were not stated. In my own case hoarseness and pain radiating from the throat to the ears, without preliminary symptoms, appeared about 17 days after operation.

Clausen's patient, soon after the diagnosis was made, coughed up a pea-shaped tumor and blood clot, with little

organization; thereafter the larynx appeared normal. In the remaining cases surgical removal was necessary. Cohen removed the growth only partially but did not have to complete the stage procedure because the remainder disappeared voluntarily. Kearney removed the growth in toto, after a delay to permit it to become pedunculated, and had no recurrence. In Gould's and in Smiley's cases secondary removal was necessary, as it was in my own case on the side on which the growth had not become pedunculated. The description suggests that in Smiley's case the (unilateral) growth was also sessile.

There seems little doubt that in all of these cases, whether or not there was a history of trauma, the granuloma originated in a traumatic ulcer of the cord, which exposed the superficial cartilaginous vocal process of the arytenoid. Kearney pointed out that such an ulcer probably heals slowly from the periphery, providing for the lesion a smaller and smaller base until eventually a pedicle is formed. He suggested that in Clausen's case, in which the tumor seems to have been coughed up, healing may have progressed to the point of self-amputation.

I have no doubt that in my own case the tube used for endotracheal anesthesia at tonsillectomy, a Magill tube No. 35, was too large. Since this experience I have made it a rule to permit the use of no tube larger than No. 32. Obviously the tube should be smooth and sufficiently flexible to permit nontraumatic insertion, while at the same time stiff enough to maintain an airway. It should never be introduced except under direct vision, with the larynx completely exposed.

The experience in this small number of cases suggests that if a granuloma of the larynx develops following endotracheal anesthesia, it is wise to defer its removal untill it has become pedunculated, otherwise, a large raw area will be left and recurrence is likely. In my own case the raw area following removal of the sessile growth on the left was commented on in the operative notes. On the other hand, a patient known to have a granuloma of the larynx should be warned that respiratory difficulties may develop and should be told to return promptly for treatment if they occur.

SUMMARY.

A case is recorded of bilateral granuloma of the larynx following endotracheal anesthesia. The five cases previously recorded are briefly discussed. Although this is an apparently infrequent sequela of endotracheal anesthesia, precautions designed to prevent it should be taken in all cases.

REFERENCES.

1. LUNDY, JOHN S.: Clinical anesthesia. A Manual of Clinical Anesthesiology. Philadelphia and London, W. B. Saunders Company, 1942.

2. CLAUSEN, R. J.: Unusual Sequela of Tracheal Intubation. Proc. Roy. Soc. Med., Sect. Anesthetics, 25:1507, 1932.

3. GOULD, R. BLAIR: Laryngeal Granuloma Following Intratracheal Intubation. Brit. M.J., 2:499, 1935.

4. COHEN, M.: Tumor of Vocal Cord Following Nasal Endotracheal Anaesthesia (Clinical Memoranda). Brit. M. J., 1:283, 1938.

5. SMILEY, W. A.: Polypoid Granuloma of the Larynx Following Endo-Tracheal Anesthesia. Ann. Otol., Rhin. & Laryng., 49:556, 1940.

6. KEARNEY, H. L.: Bilateral Granuloma of the Larynx Following Intratracheal Anesthesia. Ann. Otol., Rhin. & Laryng., 55:185, 1946.

BOOK REVIEWS.

Ear, Nose and Throat Symptoms—Diagnosis—Treatment. By George D. Wolf, M.D., Assistant Clinical Professor of Otolaryngology, New York Medical College, New York. Five hundred twenty-three pages including Index and 145 illustrations, many of them in colors. Philadelphia: J. B. Lippincott Company, 1947.

A great deal of time and effort on the part of many people is required in the production of a book. It seems a pity that time and effort were wasted in the production of this book. In his preface the author states that his book "is intended for students, teachers and practitioners who desire a more extended, as well as a more advanced, view of the subject than the novice does, and yet do not wish to become enmeshed in a welter of details." One wonders how a student may obtain more advanced knowledge of a subject without going into details. The author, after his preface, opens his book with the following profound observation: "An emergency is an event or combination of events or circumstances calling for an immediate action. Only too often, however, the physician's time and effort are wasted on a false alarm because of a nervous family that cannot differentiate superficially alarming symptoms from a real emergency in which the life of a patient depends upon prompt help. Such critical situations are fortunately the exceptions, but when encountered must be met by the attending physician with alertness, calm, good judgment and quick action. A pause for sufficient time to map out a plan of campaign will prove eminently worthwhile. The proper medicaments, packing material and instruments ready for immediate use must be at hand before treatment is attempted. Good light is indispensable in dealing with urgent otolaryngologic problems. Speedy, yet deliberate action, is the order of the day." We assume that this paragraph was written specifically for those desiring a more extended as well as a more advanced view of otolaryngology. The book continues in the same vein of profundity through some five hundred pages. For example, "Peritonsillar abscess is a deep seated infection of the tonsillar tissue with pus formation in the tonsils are peritonsillar tissue." Again, "Polyps are the most common tumors of the nose. They are not true tumors but myxomas of the nose, a manifestation of hyperplastic rhinitis. They may be either neoplastic or polypoid in nature." Further, "Pressure on the trachea is caused by neck infections and tumors in this region. Tumors of the thyroid, especially the substernal type, are apt to cause compression of the trachea."

There are many illustrations throughout the book, but few of them are revealing and some erroneously captioned. Some illustrations in color, which one must suppose added considerably to the cost of production, were distributed gratis to most otolaryngologists as advertising matter not long ago. There is presented at the end of each chapter a short bibliography. This would be useful if the author had chosen his references with care. The book is well printed on nice paper and it has a fine looking cover. We feel that it does not fulfill the author's intention and certainly could never take the place of standard textbooks of otolaryngology.

T. E. W.

Hearing and Deafness. A Guide For Laymen. Edited by Hallowell Davis, M. D., Four Hundred Ninety-Six pages with Index and several illustrations. Forewords by Louise Tracy and the Editor. New York: Murray Hill Books, Inc., 252 Madison Avenue. 1947. Price $5.00.

A general survey of the field of information which has been accumulated regarding hearing and the management of hearing impairment and deafness. It is expressed in terms understandable to the layman and, therefore, is a guide to the deafened and those with whom they are in close contact, yet is sufficiently comprehensive to be a useful reference for all those engaged in some phase of the education and rehabilitation of the deafened.

The book is organized in six sections. The first presents an overall view of the objectives, the plan of organization and a statement of contents of each of the following sections.

The next three chapters which deal with the physics and psychology of hearing, the structure and physiology of the sense organ and the medical and surgical aspects of prevention and treatment of deafness provide the basic information which is necessary to supplement the advice of the otologist and to supply the answers to a multitude of questions which must be settled preparatory to the adoption of any type of mechanical aid to hearing or long-term program of education to overcome the disability.

The next section, devoted to the various tests of hearing, the mechanical details of hearing aids, their selection, use and care provides information of a general and specific nature which is of great value as a reference both to those concerned with giving advice and to the prospective purchaser who must learn to use the instrument. It should do much to aid in the selection as well as prepare the user for the problem of learning to get the most value out of a hearing aid.

A chapter is devoted to speech reading (lip reading), conservation of speech and auditory training, and the organization of rehabilitation centers during the recent war.

The following sections present the history of progress in education of the deafened, the development of a more enlightened social viewpoint towards the problem and to the various aspects of training as guidance as they are now becoming generally available.

They discuss the training of deaf children both in the home and in schools and stress the psychological aspect of the problem as it affects the child, the parents, or the deafened adult.

The final chapters provide information regarding organizations for the aurally handicapped, employment for the hard of hearing and vocational guidance.

The book should fill a great need not only as a reliable source of information for the deafened but also as a reference and guide to all those who in any way are concerned with the alleviation of his disability.

J. R. L.

IN MEMORIAM

GEORGE ELMER SHAMBAUGH, M.D.,

1869-1947.

George Elmer Shambaugh, for many years Professor and Head of the Department of Otolaryngology of Rush Medical College, Chicago, died at the Wesley Memorial Hospital November 30, 1947 of cancer of the lung.

Born on November 15, 1869 in Iowa, Dr. Shambaugh studied medicine and graduated from the University of Pennsylvania, Department of Medicine, Philadelphia in 1895. Following this he spent two years in post-graduate work at the Universities of Berlin and Vienna. He then returned to Chicago and in 1900 became an Instructor in the Department of Anatomy of The University of Chicago and during the next several years carried on research as well as the practice of otolaryngology. His investigations on the anatomy and histology of the internal ear during the ensuing years gained him international recognition. In the year 1912 he became the first American to be awarded by an international committee the Baron de Lenval prize for research on the internal ear.

Dr. Shambaugh was otologist and laryngologist to the Presbyterian Hospital from 1902 to 1935. In 1916 he became Professor and head of the Department of Otolaryngology at Rush Medical College, retiring in 1935 to become Clinical Professor Emeritus.

He was a member of numerous medical and scientific societies, including the Chicago Medical Society, the American Medical Association and the American College of Surgeons. He was secretary of the Section of Laryngology and Otology of the American Medical Association from 1908 to 1911, Chairman of the Section in 1911-1912 and a member of the

House of Delegates in 1916. He was also a member of the American Laryngological, Rhinological and Otological Society, Chicago Laryngological and Otological Society, Institute of Medicine of Chicago, The American Otological Society and the American Laryngological Association. His eminence in his special field gained for him the distinction of having been president of the latter four societies. Dr. Shambaugh was one of the founders and later an honorary member of the American Board of Otolaryngology. He was the editor in chief of the Archives of Otolaryngology from the time of its establishment in 1925 until 1937. At that time the Board of Trustees of the American Medical Association tendered to him its appreciation for his contributions to the advancement of the Archives.

During his many years of active practice and scientific work Dr. Shambaugh contributed many articles to current medical literature and wrote chapters for several textbooks in his special field. He achieved world-wide recognition for his scientific attainments and was at all times an outstanding leader, an influence for improvement in postgraduate training in the specialty and for the adoption of sound medical principles in the practice of otolaryngology.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

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Maico Type K; Maico Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Microtone T-4 Audiomatic.

Manufacturer: The Microtone Co., 4602 Nicollet Ave., Minneapolis, Minn.

Otarion Model A-1; Otarion Model A-2; Otarion Model A-3; Otarion Model A-4 Jr.; Otarion Model E-1.

Manufacturer: Otarion Hearing Aids, 448 N. Wells St., Chicago, Ill.

Paravox Models VV2 and VV3; Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS.

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Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

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The coming Section Meetings will be held as follows:

Eastern Section—Jan. 16, 1948.....New York, N. Y.

Middle Section—Jan. 19, 1948.....Columbus, Ohio

Southern Section—Jan. 23, 1948.....New Orleans, La.

Western Section—Jan. 31-Feb. 1.....San Francisco, Calif.

(The mid-winter Council Meeting will be held in New York, N. Y., on Jan. 17, 1948.)

Arrangements have been made to hold the Annual Meeting at Chalfonte-Haddon Hall in Atlantic City on April 7, 8 and 9, 1948. The mornings will be devoted to scientific sessions; the afternoons will be left free.

The American Otological Society and the American Laryngological Association are planning to hold their meetings at Old Homestead, Va.—the former on April 12 and 13, 1948; the latter on April 14 and 15, 1948.

This plan to have a three-day Triological meeting with free afternoons and an interval of two days between the Atlantic City and the Old Homestead meetings is projected in order to relieve the strain of concentrating within one week the combined activities of these three societies. Address any inquiries to Dr. C. Stewart Nash, Secretary.

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Meeting: New Orleans, La., Jan. 23, 1948.

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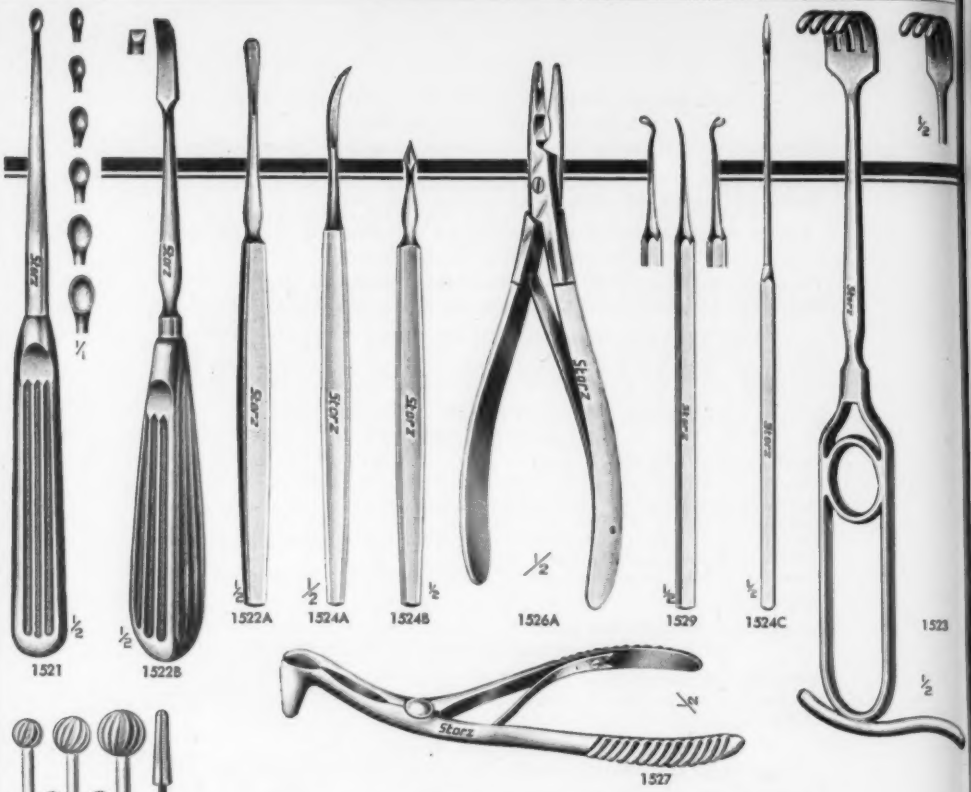

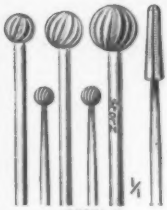

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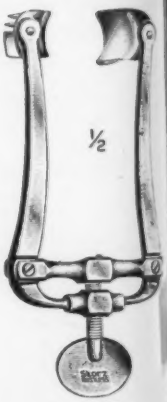

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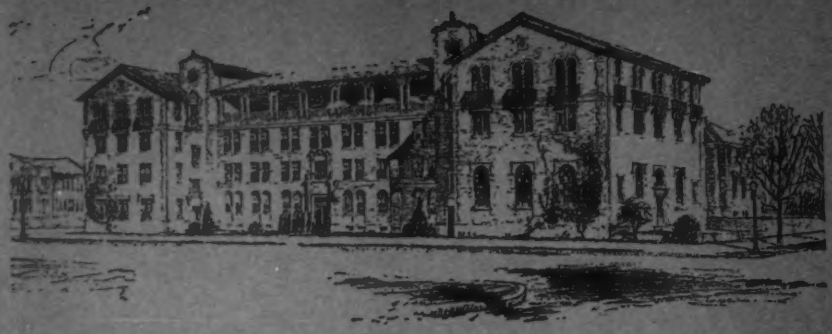

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